VOL. XXI., NO. 7 MAY, 1928.

Medical Lib.

PROCEEDINGS of the ROYAL SOCIETY OF MEDICINE

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> SIR ALMROTH E. WRIGHT, M.D., Lancet, Dec. 14, 1927.

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PROCEEDINGS

OF THE

ROYAL SOCIETY OF MEDICINE

EDITED BY

T. WATTS EDEN, M.D.

AND

HUGH THURSFIELD, M.D.

UNDER THE DIRECTION OF

THE EDITORIAL COMMITTEE

VOLUME THE TWENTY-FIRST

SESSION 1927-28

PART II

MAY TO OCTOBER

LONDON

LONGMANS, GREEN & CO., LTD., PATERNOSTER ROW

1928

Medical Soc.

Section of Anæsthetics.

President—Dr. CECIL HUGHES, O.B.E.

[*March* 2, 1928.]

JOINT MEETING WITH THE

Section of Obstetrics and Gynæcology.

DISCUSSION ON ANÆSTHESIA IN OBSTETRICS.

Mr. EARDLEY HOLLAND (Section of Obstetrics).

THE special requirements of anæsthesia in obstetrics may be stated as follows:—

(1) The method must be simple, safe, and capable of being used by those who have had no particular training in anæsthetic methods.

(2) The pain must be abolished, or much diminished in severity, over long periods of time; this is a peculiar aspect of the problem, because labour may last for twenty-four or forty-eight hours or more.

(3) The force and frequency of the uterine contractions must not be diminished.

(4) The life of the fœtus must not be endangered.

The subject has two aspects. Firstly, the administration of anæsthetics to relieve the pain of labour; secondly, the administration of anæsthetics for obstetrical operations, such as forceps, version and Cæsarean section; this aspect, for the purpose of this discussion, seems far the less important of the two.

Anæsthesia for Obstetrical Operations.

The most suitable anæsthetics for these operations are those for operations in general, and are, in individual cases, governed by the same considerations. I believe that chloroform and ether are equally safe for the fœtus, with the reservation that prolonged deep anæsthesia with either is unadvisable, especially if the vitality of the fœtus has been depressed, or is likely to be depressed, by such unfavourable influences as prolonged labour, cranial pressure or maternal hæmorrhage, or by the administration of morphia to the mother shortly before delivery. I am aware that the statement that, for the fœtus, ether is as safe as chloroform is to some authorities provocative; and I hope it is a point that will be discussed by those who hold the contrary view. The fœtus is very sensitive to morphia. Morphia should not be given within two hours of the birth, owing to the risk of fœtal apnœa from the action of morphia on the fœtal respiratory centre. Morphia and chloroform or ether have a cumulative effect on the fœtus. Stovaine-spinal-anæsthesia I mention merely to praise it for Cæsarean section for certain indications (notably for cases complicated by diabetes, cardiac disease, goitre, and severe ante-partum hæmorrhage), and for its remarkable effect on the uterine muscle, whereby Cæsarean section usually becomes almost a bloodless operation.

The Pains of Labour.

It is not disputed to-day by anyone that every woman should be spared the pain of labour, especially when the means to that end are so safe, simple and easy of application. The first anæsthetic ever given during labour was given by Sir James Young Simpson, almost exactly 81 years ago. Just as he wrote the first words on the subject, so he may be said, metaphorically, to have written the last; for after one has read his writings, published during the years 1847 and 1848,[1] one is compelled,

[1] "The Obstetric Memoirs and Contributions of James Y. Simpson," edited by Priestly and Storer, 1861, xi, 578-721.

in sheer admiration, to admit that, both from the humanitarian and from the scientific point of view, there remains very little to add. The method of "intermittent chloroform administration" which he devised is, as regards technique, scope and limitations, such as is used to-day by the majority of obstetricians. In fact, the only improvement may be said to be the apparatus used, namely, the inhaler introduced by Junker in 1867.

In private practice, anæsthesia or analgesia is universal. In hospital practice in this country, both in the maternity ward and "on the district," little, if anything, seems to be done to relieve pain. The greatest problem of all is presented in the practice of midwives. If practising midwives cannot use this means for alleviating the pains of labour, about 75 per cent. of the women of this country must go on suffering. This is a problem which surely is not incapable of solution. If only midwives could relieve the suffering of their patients, no doubt they would at once attain the position they deserve in the midwifery service of the country. There is not the least objection to their giving analgesic drugs such as choral and opium; but, even if a sufficiently simple and safe apparatus could be devised, I hesitate to go so far as to suggest they might be allowed to give more potent anæsthetic drugs.

The requirements of anæsthesia or analgesia for the first and second stages are very different. The pain of the first stage, which is due to the contraction of the uterine muscle, is less severe than that of the second stage, during which the pain from stretching of the vulval orifice and perineum is superadded to the pain of uterine contraction. There can surely be no pain more agonizing than that which, in a primipara at all events, accompanies the expulsion of the child, associated as it is with extreme distension and laceration of a very sensitive orifice. Simpson, who had ample experience of the pre-anæsthetic days of surgery, said that "the degree of actual pain usually endured during common labour is as great as, if not greater, than that attendant upon most surgical operations."

The pain of the first stage, even though it cannot be abolished, can at all events be made bearable by analgesic drugs, such as choral, morphia and hyoscine; but during the second stage, and certainly during the actual expulsion of the fœtus, general anæsthesia is required. Although most women suffer severely, a small proportion suffer no pain worth speaking of, no more indeed than an uncomfortable aching during each uterine contraction. In this connexion, I wish to emphasize the effect of emotion—that is, of fear, anxiety and apprehension—upon the sufferings of a woman in labour. In a state of confidence and calm, the suffering is less intense than in a state of mental and physical tension.

The fact that a woman knows that she will be relieved of pain by anæsthetics, goes a long way to removing "the state of anxiety and dread of anticipation which silently annoys and haunts her" (Simpson).

The Method of Anæsthesia in Common Use.

The only method of anæsthesia which I shall speak about is that in common use, namely, intermittent chloroform.

Chloroform should not be given until the second stage has begun, or, at all events, not until the first stage is nearing its end. Otherwise the uterine contractions will be diminished in force and frequency. It is not really a disadvantage of the method that it cannot be used earlier in labour, because the pains of the first stage can be satisfactorily relieved by analgesic drugs.

As soon as it is perceived that the uterine contractions are badly borne and begin to cause suffering, a dose of chloral hydrate or an injection of morphia and scopolamine is given. The time for this varies much in different cases, and no rule can be followed. Chloral is a wonderful drug which has undeservedly gone a little

out of fashion. It should be given in a large dose (I usually give 30 grains), and should be taken in sips in a tumblerful of water, otherwise the patient may vomit. Instead of chloral, or subsequently to chloral, if the first stage is prolonged and the chloral effect wears off, morphia and scopolamine may be given. The precaution must be taken not to give morphia within two hours of the birth of the child, because of the risk of fœtal apnœa.

As soon as the second stage of labour is reached (or sooner than this in multiparæ) chloroform is given from a Junker inhaler. It is very seldom that patients refuse chloroform, but even nowadays there are women who desire to remain perfectly conscious during the birth of the child.

The method of administration of chloroform during labour is a special one. It cannot be expected to come by intuition; instruction and experience are necessary before it can be used to the fullest advantage. Now that medical students are taught in in-patient maternity departments there is ample opportunity for learning the method, but it is doubtful if they ever see it used in cases of normal labour; this is a point which should receive the attention of teachers of obstetrics. Almost everything there is to state about the use of chloroform in labour has been stated with the utmost clearness by Simpson. In general, there are two main difficulties: (1) to keep the patient in a state unconscious, or barely conscious, of pain, and (2) not to have her so deeply anæsthetized that the uterine action is interrupted. These two objects are attained by getting the patient into the first or "light" stage of anæsthesia. Once this stage has been reached it can be sufficiently maintained by working the inhaler during each recurring uterine contraction. When the contraction is over the chloroform is withdrawn, and is only again administered when the movements of the patient, or her requests for "more chloroform, please," or the consistency of the uterus, indicate a recurring contraction. The Junker inhaler is a simple and safe apparatus, which can be operated by the nurse or by a lay person present at the labour, or even by the patient herself. The degree of anæsthesia thus attained is ample, and only in the rarest cases are the force and frequency of the uterine contractions diminished. Nor are the reflex bearing-down efforts weakened; indeed the common effect is that the progress of labour is hastened, because the tendency to contract the levator ani muscle, and thereby hold the advancing head off the painfully distending perineum, is abolished.

The abdominal and levator ani muscles are antagonistic; under light anæsthesia, however, because pain, and the dread of the child suddenly bursting forth into the world, are relieved, those opposing muscles act in perfect harmony, powerful bearing-down efforts being associated with relaxation of the pelvic floor.

As the head is passing through the vulval orifice, which is the time of greatest agony, the degree of anæsthesia must be deeper.

I have had very little experience with light ether anæsthesia during labour. It is believed by some to have a more stimulating effect than chloroform on the uterine contractions. But the advantages of chloroform over ether in ease of administration, speed of action, pleasantness and portability, are so great that it seems likely to maintain its popularity.

Other Methods.

Of these I have no experience. From all reports, gas and oxygen anæsthesia seems likely to possess many advantages over the chloroform method and may prove to be the most effective method yet devised for obstetrical anæsthesia.

26 Blomfield—Gilliatt: *Anæsthesia in Obstetrics*

Dr. J. Blomfield (Section of Anæsthetics)

said the first thing that struck an anæsthetist in connexion with obstetric cases was that, whereas in all other respects the modern gynæcologist considered a labour was a surgical occasion, his attitude as regards anæsthetics was quite different. The surgical technique was regulated as carefully as for any other operation, but, whereas at other operations the gynæcologist would not tolerate conditions in which his patient endured pain or in which the anæsthesia depended on himself or on some unskilled person, these conditions were accepted as normal for confinements. It was surely, in these days of falling birth-rate, a matter of importance that there should be nothing to deter childbirth, and the dread of pain was one of the deterrents. It *could* be abolished, and a new tradition of painless childbirth should be propagated. Women need suffer no more pain through labour than through any ordinary surgical operation. For this to be brought about the anæsthetist must be early on the scene. He was perfectly willing to come if the gynæcologist summoned him. The means at his disposal for ensuring painless labour were varied, and he (Dr. Blomfield) confined his remarks to the management of normal patients. The ideal arrangement was the administration of a sedative, omnopon or heroin, or scopolamine and morphia, early in the first stage, and the frequent intermittent use of "gas and oxygen" as soon as the pains caused inconvenience to the patient. The face-piece was to be applied directly a pain started. For the first few breaths nitrous oxide was given alone and then diluted with the oxygen in various percentages according to the patient's requirements as the anæsthetist found them. Usually it was necessary to reinforce the gases with a little ether when the head was stretching the perineum to the utmost, and real surgical anæsthesia was brought about if the gynæcologist desired it. The practical drawback to gas and oxygen was that it was rarely possible to use it outside nursing homes or hospitals owing to mechanical difficulties. There must be a narrow bed and suitable apparatus. When it could be used it was the most desirable anæsthetic, because it interfered less than any other with the metabolism of both mother and child. Apart from mechanical difficulties, however, there were occasional patients for whom it was inefficient.

The next anæsthetic to be considered, chloroform, was invariably successful so far as keeping the patient quiet and free from pain was concerned. Moreover, properly used, it need not prolong labour. Its use must be meticulously regulated, a very weak vapour being employed, and a state of analgesia rather than anæsthesia being maintained until the accoucheur wanted relaxation and absence of effort on the patient's part. The objections to chloroform as a routine anæsthetic in ordinary surgery do not apply to obstetrics. There is for one thing a complete absence of that real but ill-explained danger from psychic effects in the early stage of administration to a frightened person. The woman in labour, so far from fearing the anæsthetic, is crying out for it. With regard to more recently introduced methods, Dr. Blomfield said that "synergistic anæsthesia" did not appeal to him. He believed the rectum to be an inappropriate route for the introduction of anæsthetics in a woman whose pelvis was already over-filled. He had tried ethylene and acetylene and thought their disagreeable smell more than counterbalanced their advantages, which were analogous but inferior to those of nitrous oxide gas.

Mr. W. Gilliatt

said that in those cases which were attended by a doctor, assisted only by one person, who was sometimes qualified and sometimes not, the anæsthetic of choice and convenience was chloroform. Chloroform had the disadvantage of diminishing the strength and frequency of the uterine contractions to a greater extent than

ether did, but it was more popular because it could be given with safety and efficiency by a Junker's bottle connected by tubing to a mask instead of a mouthpiece.

The presence of an anæsthetist enabled the patient to be given any anæsthetic thought desirable, and he personally had no doubt that, for the relief of pain, the gas and oxygen mixture was the best anæsthetic for many reasons. It was usually necessary to give ether by the open method for a short time during the actual delivery of the child.

He referred to the dangers of delayed chloroform poisoning, of which he had seen five cases and of which he gave the details. The feature in all of them was that they had had two anæsthetics at short intervals, i.e., twelve hours to five days, and that on each occasion the second anæsthetic had contained chloroform, either pure or in mixture. He strongly emphasized the danger of using chloroform as an anæsthetic in midwifery cases, owing to the difficulty of being sure that a second anæsthetic might not be necessary.

Dr. H. Richards.

As an anæsthetist I propose only to deal with the selection of the anæsthetic to be used in obstetrics, which appears to depend entirely on the state of the patient. Is the patient to be examined under an anæsthetic to see whether the child will come through? Is her labour to be induced? Is she in labour? and has something gone wrong which may want manipulation or forceps, and, therefore surgical anæsthesia?

It does not matter in the least what anæsthetic is chosen when the patient is in labour, provided she has not had a previous anæsthetic; if she *has* had a previous anæsthetic nothing that contains even a trace of chloroform should be used, as it is the repeated small doses of chloroform, spread over a time, that do the damage, as has been shown in the cases referred to by Mr. Gilliatt.

My routine method in cases in which more than one anæsthetic may be required is to use a Hewitt wide-bore inhaler, and the anæsthesia is begun with gas, a preliminary injection of $\frac{1}{100}$ gr. of atropine being given half an hour beforehand. By this method any degree of anæsthesia can be reached with comparative safety for mother and child; should a second anæsthetic be required the same method may be used, but, as frequently happens, labour has been going on for some time and the patient is partially anæsthetized, either by herself through a Junker, or, by the obstetrician, on a mask. This type of case does extremely well with ether on a mask, a light anæsthesia being very easy to maintain without unduly depressing the contractions of the uterus.

Nitrous Oxide.—Nitrous oxide alone, or nitrous oxide and oxygen, is used in cases in which the patient is always violently sick after any anæsthetic, although, personally, I think patients are just as sick after gas and oxygen as they are under any anæsthetic; the psychological factor may come in after gas and oxygen is used, because patients have an idea that they are less sick after gas and oxygen than after anything else.

I use nitrous oxide alone when a patient is unduly apprehensive of pain, or is unwilling to endure any at all. The apparatus may not be familiar to all present and I will describe it in some detail. It is put up by Coxeters on the same lines as the American one, the idea being that the patient shall give herself gas over a long period. The cylinder is fitted with a pressure reducer, which contains a diaphragm adjustment. The delivery tube at its distal end has a screw pressure adjustment, from which radiate a couple of concave metal arms, which pass up and impinge on the gas bag at its middle. These are connected with one another by a rubber band, which passes round the circumference of the bag and is taut when the bag is full

of gas; any pressure can be obtained in the bag by an adjustment of the screw attachment—the tighter the screw the greater the pressure. The flow of gas from the cylinder is controlled by the diaphragm in the pressure reducer on the cylinder head; as the pressure is reduced in the bag the gas is liberated from the cylinder, until the bag pressure is raised to a point where the diaphragm is depressed and the flow of gas is stopped. It is therefore possible to leave the cylinder turned on when not in use, and the patient, in theory, is able to give gas to herself when a "pain" starts. The gas reaches the patient by an attached valve, which is shielded by a wire cage, admitting a finger. The valve is operated by depressing it, and when the finger is relaxed the valve closes itself automatically, thereby shutting off the gas. The cage over the valve is to prevent it from being interfered with by bedclothes, pillows, etc.

This apparatus is excellent in theory, but I do not think it works in practice—on more than one occasion I have been with a patient and let her use it herself, and I have found that the finger on the escape valve becomes stiff when consciousness is lost, so that the valve is not closed and the patient becomes cyanosed.

My own method of using it is as follows. When the patient feels the onset of a pain, she indicates it to me by raising a finger. I then open the valve and continue to give gas without any oxygen, the valve being opened in the ratio of 3 to 1, and this is continued until the pain ceases, a point which can be ascertained by the change in the respiration: there is no cyanosis and very little salivation. In my experience there is no sickness, although I have given gas to patients for over four hours. The great advantage in the method is that patients are extremely well afterwards and the contractions of the uterus from being lessened are, if anything, increased. The disadvantages are the time taken, the amount of gas used, and the cumbersome apparatus. I stop the gas as soon as the head is on the perineum, and keep the patient lightly anæsthetized with open ether; I do not use gas-oxygen-ether sequence because it is not wise to have rebreathing over what may be a considerable period.

Cæsarean Section.—In my experience, induction with gas and ether in an inhaler, and continuance of anæsthesia with open ether, is in every way an admirable method, and surgical anæsthesia maintained thereby has given no anxiety either for the mother or the child.

Spinal Anæsthesia.—Stovaine has its place in obstetrics as in other branches of surgery, particularly in cases of failing heart, in which the patients appear to do well with a small dose—0·4 c.c. of a 10 per cent. solution of stovaine in saline. Having found that the stovaine is working efficiently, it is advisable to cover the patient's eyes and give no general anæsthetic at all, because, in my experience, whatever anæsthetic is chosen, it invariably stirs up some lung complication which may tip the balance against the patient. There seems to be no difficulty in getting the uterus to contract when once the baby is delivered, the uterus becoming extremely firm at once.

Mr. H. Edmund Boyle

said that during his visit to America as a representative of the Section, he had been particularly impressed by the work done by Dr. Wesley Bourne, of Montreal, in the administration of gas and oxygen, especially for the performance of Cæsarean section. It was not until some time after his return that he (the speaker) could persuade obstetricians to allow him to use the mixture in this country for midwifery cases. With this anæsthetic, a short time after the patient's confidence had been obtained, a state of analgesia was produced in which the patient would bear down and do other

things on request. He did not believe the "pains" and propulsive efforts were diminished by the anæsthetic, but thought they were actually increased in strength and frequency. Patients were glad to have been practically conscious during the whole time. If the woman bore down well the whole time, the length of the labour was shortened—an important matter. Just as the head was being born he always gave ether; it kept the patient quiet, and so lessened the probability of the perineum being torn by a sudden expulsive effort. As soon as the child was born he cut off the gas, and gave a mixture of carbon dioxide and oxygen for a few minutes; the mother and child were then noted to breathe more deeply, and assume a natural pink hue. Gas and oxygen alone could then be continued until the birth of the placenta, after which no further anæsthetic was required.

With regard to the use of apparatus being inconvenient in private houses, he had arranged with a firm to send round to a house the apparatus required, for a small charge, so that when the anæsthetist arrived everything was in readiness. He regarded the gas and oxygen mixture as the best anæsthetic for midwifery, and he hoped its use would become more general in this country.

Dr. H. Beckett-Overy.

Before dealing with the subject, in which I am particularly interested, I may say that I purchased an automatic gas and oxygen apparatus, but found it unsatisfactory; on one occasion the patient had quite serious apnœa, giving several minutes' anxiety.

Some of us have to deal with patients not for weeks, but for months and years after a confinement. Many women suffer severely from the shock of an uncomplicated confinement, not to mention a tedious or complicated one. They are terror-stricken at the thought of another experience such as the last.

If there are safe means of anæsthesia, it is our bounden duty to avail ourselves of them in all cases, even if it entails more supervision and a greater responsibility. That there are safe means which can be easily carried out by a doctor and nurse working alone I unhesitatingly assert, and no woman should be allowed to go through the actual birth of a child unanæsthetized.

Like Dr. Holland, I use a Junker's apparatus with pure chloroform. It is furnished with pieces of tubing at least 3 ft. in length, and the bottle is hung on the head of the bed or against the wall. In this position it is readily available to patient, nurse and doctor. In multiparæ, when the os is dilating rapidly or when the second stage has been reached on arrival, the patient is shown how to use the apparatus and is trusted to squeeze the bulb immediately each pain is about to begin. She usually succeeds in rendering herself partially unconscious in a few seconds, and as she becomes more unconscious she relaxes her efforts and so, if a moderate injection of hyoscine and morphia has preceded at an earlier stage, sleeps till the next pain. In this way the majority of patients are enabled to reach the final stage with immense relief and a great saving of nervous and physical strength.

When the head begins to crown, the nurse is directed to take charge of the bulb. The accoucheur then supervises the administration himself, so as not to abolish the abdominal contractions entirely, but as the head is about to be born the amount of chloroform is vigorously increased, ceasing immediately the head is born. In the majority of cases the patient has little recollection of the second stage, and is entirely oblivious of the actual birth.

In the case of primipara or multipara, where the os is not dilating quickly and the patient is becoming intolerant of the pains, $\frac{1}{6}$ gr. omnopon and $\frac{1}{150}$ gr. hyoscine are given. In the majority of cases this does not retard, but often quickens,

dilatation. The dose of hyoscin may be repeated, but no attempt to obtain complete amnesia, or what has been called "twilight sleep," by repeated doses is made. There is a danger of difficulty in getting the child to breathe after this practice.

With the above described routine there are far fewer complications, both immediate and remote. The patients eat, sleep and nurse better, and temperatures are practically non-existent (although this fact is also due to the practice of allowing patients to sit up in bed from the first day, for at any rate a short time). Whatever delay there is, and there is some delay, is more than compensated for by the relief of the patient and the saving of the perineum. Many lacerations occur through too rapid expulsion, and one can conceive of nothing more cruel than retarding the passage of the head with the patient fully conscious.

It is little short of a scandal that nearly a hundred years after Simpson's introduction of chloroform it is not used as a routine in lying-in hospitals and maternity wards.

Mr. Harold J. Malkin

spoke of the synergistic method, i.e., administration of morphia and magnesium sulphate intramuscularly, followed by the rectal instillation of ether and quinine in olive oil, as used by Gwathmey, in New York.

The American view of it was that only a small dose of morphia was necessary, and that the magnesium sulphate kept the action more effective and over a longer time. The quantity of ether needed was also small, and it was said that the administration could be rendered so automatic that the constant attention of the obstetrician was not required.

He had used the method in twenty cases at University College Hospital, and twelve of these were entirely successful, i.e., the patient slept between the pains and stirred only with the pains. In some cases the woman had not known when the child was born.

In one case there was excitement, in two the method failed completely, in one he gave the morphia rather late, the second stage supervened sooner than he expected, and the child was born blue. In two cases he began administration too early and apparently delayed the first stage some hours.

Now that he was better acquainted with the technique he felt certain of securing a large percentage of successful cases with the method which, he was convinced, possessed advantages.

Dr. G. Clark

said that in a total of 3,000 midwifery cases without a single maternal death during a long period of years, in which 15 per cent. required an anæsthetic, pure chloroform was always used with success.

Even in a series of major surgical operations, the same anæsthetic was used with satisfactory results.

Dr. Z. Mennell

stated that as he had seen both Dr. Bourne's and Dr. Mackesson's work in America, he would like to corroborate what Mr. Boyle had said with regard to its excellence and to the good results obtained by their use of nitrous oxide and oxygen in ordinary uncomplicated labour. But—and there was always a but in such work—conditions on the other side of the Atlantic were quite different to those existing here. The women, rich and poor, were all confined in hospital, and there was always a gas and oxygen machine handy with thousands of gallons of gases—standing 6 ft.

high and more. There was nothing portable about them, and they would require a pantechnicon van to take them about. Mackesson made a portable machine, but even that was bulky and not suitable for a long case without change of cylinders.

Dr. Mennell went on to say that he considered, under the conditions prevalent in this country, that chloroform given properly and to the obstetrical degree only was the best and most efficient anæsthetic we had in this class of case; in fact, he considered that it was only in this work that the use of chloroform was justified as a routine. Ether was of course satisfactory, but it necessitated the use of preliminary medication and much greater skill, which was not always obtainable for its successful use. After all, the condition known as "delayed chloroform poisoning," really an acidosis, was recognized also as occurring after ether anæsthesia, and even without an anæsthetic at all. It was not fair to chloroform to attribute all such cases to its use. Perhaps some of the cases reported to-night might have occurred with any other anæsthetic, as their carbohydrate metabolism had been interfered with. On the whole, he considered chloroform to be the anæsthetic of choice to relieve the pain of ordinary uncomplicated labour.

Section of Balneology and Climatology.

President—L. C. E. Calthrop, M.B.

[*March* 8, 1928.]

The Value of Marine Health Resorts, with Special Reference to Children.

By R. Fortescue Fox, M.D.

Many years ago Sir Herman Weber pointed out that no country could compete with Great Britain in variety and value of marine health resorts. He expressed surprise that, although England was the first country to establish seaside hospitals and sanatoria, we had done comparatively little to develop the facilities for treatment on the coast, whilst Continental countries had been very active in following our lead.

Häberlin, an authority on sea bathing at Föhr, an island on the other side of the North Sea, in a recent article[1] traces the history of modern thalassotherapy to the work of Richard Russell in 1750. "This English physician," he says, "invented the sea." The internal and external uses of waters were studied by many medical men of distinction in England in the eighteenth century,[2] and the foundation of the "Royal Sea-bathing Infirmary" at Margate, in 1796, marked the opening of a new chapter in physical medicine. Meanwhile, enterprising individuals made trial of the sea for their own ailments. We are told that the first bathing machine was the invention of Benjamin Beale, a Quaker, at Margate, in 1757, and that an invalid, with a similar machine, made his appearance at Weymouth, in 1767. Interest was aroused, and finally the royal example of the Georges at Weymouth and Brighton brought the seaside into favour in England amongst those who could face the then expensive and troublesome journey. Evil times, lack of communication, apathy in health matters, long hindered access to the coast. It was not until the latter half of the nineteenth century, with the advent of railways, increasing wealth, greater care for children, and the study of medical climatology, that the movement to the sea gathered volume and became almost a national cult. The great centripetal movement of populations to the cities, which belongs to our civilization, is now a little mitigated by the centrifugal movement of parents and children from the cities to the sea. The yearly summer exodus in England alone is counted in millions. At most localities statistics are not available, but at Southend-on-Sea, for example, the number runs into hundreds of thousands, and reports of sea bathing at Blackpool, Margate, Brighton, Hastings, Weymouth, Bridlington, Yarmouth, Scarborough, all tell the same tale. The same is true of Continental and American resorts. We hear of many thousands on the coast in New South Wales, and a recent railway report from the Argentine mentions extensions necessary beyond Mar del Plata to the fine sands and clean ocean waters at Miramar, Necochea and Claromeco, to meet the constantly growing seasonal traffic.

Ord, long ago[3] wrote of our instinct to seek maritime surroundings, and of the moral title of islanders to the uses of the seaside.

1 *Archives of Medical Hydrology*, August, 1927.

2 In addition to Russell's work ("A Dissertation on the Use of Sea-water, in the Diseases of the Glands," translated from the Latin of William Russell, M.D., 3rd ed., London, 1753), the following may be mentioned: T. Glass, "Ancient Baths and their Use in Physic," 1752; John Rutty, "Mineral Waters of Ireland," 1757; T. Short, "General Treatise on the Cold Mineral Waters of England," 1766; C. Lucas (1713-71), "An Essay on Waters," in 3 parts (in the R. C. P. Library); Donald Munro, "Mineral Waters," 1770; William Saunders "Mineral Waters and the Use of Cold and Warm Baths," 1805.

3 "Climates and Baths of Great Britain,"

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All medical men and social workers must welcome this movement to the sea. Such a movement may well become vitally important for the populations in our crowded cities. It has within it, as Theodore Williams remarked at one of our meetings, great elements of pleasure and of health, but is not without problems and difficulties. Careless bathing for delicate children is a great evil. Dr. Hutchison has pointed out that acute gastro-intestinal catarrh is common after sea baths, and he has seen rheumatism developed "over and over again." Dr. Marvel, of New Jersey, was so impressed with these risks that in a paper, published in our *Proceedings*, he doubted whether sea baths did not produce more harm than good. Any summer visitor to the seaside can see for himself the injury to children of prolonged exposure to cold water, with fatigue and neglect of the most elementary precautions. At many of our resorts the arrangements are quite unsuitable for children and invalids. On the other hand, at some of our leading sea-bathing stations, such as Margate, admirable provision is made of bathing pavilions, hot drinks and hot foot-baths for bathers leaving the water.

In spite of all drawbacks the popular and the medical recourse to sea air and sea baths is abundantly justified, both in summer and in winter. I am a firm believer in the "blue-water school" in medicine. The sea is our first line of defence. So with our densely populated country the sea is a defence of the children against the invasion of disease.

Years ago, when the French *hôpitaux marins* were in course of construction, Landouzy emphasized the value of marine resorts in the prevention of children's diseases.

"In the case of this legion of children suffering from debility," he said, "rickets, lymphatism, those attacked by scrofula, this legion of degenerates and dystrophics, the children of tubercular subjects and drunkards, liable to all diseases and contagions, and those threatened with tuberculosis, a prolonged stay at the seaside performs wonders, resolving ganglionic enlargements, conferring upon all the tissues increased powers of resistance, modifying the nutritive capacity in common with all the functional activities of the organism—in a word, transforming sickly children into new beings."

To this may be added Dr. William Ewart's observation in a paper read before this Society[1] that catarrh often finds its natural remedy at the sea, which he attributes to "airing and salting," and especially to salting of the respiratory mucous membranes by crystalline particles.

That many of these good effects are produced, none of us will deny. We shall differ in attributing the benefit to particular factors in a rather complex therapeutic equation. Air, light, change of habit, salt water, wind, exposure, diet, etc., are all included in that equation. For myself, I am attracted by the theory which attributes our response to the sea to a factor in ontology—to primitive affinities lying deep in the origin of our being. Quinton, in France, has developed this idea.[2] He points out that the sea is the original medium of life, and that from the sea came the first living cell. And living cells in their own organic fluids in the human body still enjoy conditions none other than those of the sea. We know that leucocytes flourish and that some ova are fertilized in sea water.

The marine climates of England present a very interesting study. It is said that there are three coast climates in France. In the case of our own country there is a similar but more essential distinction to be drawn. Briefly, I would submit that the sea around the British Islands is not one but three seas; that the health indications

1 "The Treatment of Tuberculosis by Marine Climates," 1907.
2 "L'Eau de Mer, milieu organique," 1904.

for the coast are according to season; that the western sea with its soft Atlantic airs is the prescription for winter and spring; that the strong eastern airs are for summer and autumn; that the south coast, from the Foreland to Falmouth, is for all seasons; and lastly, that, with clearly defined exceptions, a good rule of practice is "the young to the east and the old to the west." It is not according to fact that the eastern climate is always tonic nor is the west always relaxing. On the contrary, the sedative climate of the west is, as has been pointed out years ago, for many people more strengthening than the stimulating climate of the east.

A distinguished president of the College of Physicians once told me that he had never noticed differences between one air and another: chemical analysis had shown that with trifling differences all airs were the same. On the other hand, how many people tell us that they always "feel the air" when they step out of the train? On the indented coast line of the British Islands, with health resorts having every variety of aspect, shelter and soil, there are a great many purely *local climates*, apart from the main threefold division above defined. Very often these secondary characteristics determine the use of climates for invalids. Many delicate persons, especially those with respiratory affections, are singularly sensitive to "change of air." There is a saying in France: "No one coughs at Hendaye." *Quot loci tot coeli.*

The marine health resort is the place *par excellence* for physical treatment, that is to say, for the scientific use and combination of physical energies operating upon the surface of the body. The necessity of continual physical impressions for the maintenance of health, and even life, the transformation of energy at the surface of the body, and the remedial value of external physical forces in the treatment of disease are only now beginning to be appreciated. We must include in the surface of the body the sensitive surface within the respiratory organs. Only at the seaside can so many forms of energy play upon the skin and respiratory membranes.

The influence of the sea upon vegetable and animal life is a very old observation. Gautier found 22 mgm. of sodium chloride per cubic metre of air nearly 60 kilometres inland, and the barilla plant, which depends on salt in the air, will grow in some parts of Spain as far as fourteen miles from the coast.

The effect of the marine climate upon animal metabolism was long ago studied by Benecke. He found that it had no effect upon nitrogenous waste, but materially increased assimilation. With this agrees the observation of Häberlin (*loc. cit.*) as regards children: increase of metabolism; healing of chronic disease of the respiratory organs; diminished liability to catch cold; increased immunity. He advocates a residence of from three to twelve months beside the sea, and considers that the best effects are often produced in the winter. The operation of the marine climate is not so much to heal as to activate the whole organ. Sea baths he regards as a reinforcement of the climatic influence.

It is well to remember that the seaside gives us the combined effects of light, air and water upon the human body. Exposure to the radiant energy of light means also exposure to marine air and to brusque changes of temperature. What is called "light treatment" generally includes exposure to cold. In the mountains the cold is extreme. It is less severe at the seaside, but at the sea the cooling of the body is acclerated by atmospheric currents and wind. When two causes are in operation, it is very easy to attribute to one of them the effects produced by the other. Leonard Hill and others have shown that the beneficial effects usually attributed to radiant energy may be produced by exposure of the body to the atmosphere without sunshine.[1] Hill has found that "body metabolism can be excited by exposure and bathing in the southern English summer to the same degree as by exposure to the winter Alpine

1 Girdlestone, *Archives of Medical Hydrology*, January, 1926, 111; Leonard Hill, "Sunshine and Open Air," 1925, 105, 117.

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climate." We may regard an ideal sun bath as the coincident application of heat and cold. To this is added, at the seaside, the special action of marine air and the cooling effect of winds. Hot and cold sea baths join to these surface actions additional heating or cooling of the body, and baths in the open sea supply the stimulation of foam and sprays and the shock of waves.[1]

Some writers recommend "simultaneous treatment" by physical remedies and drugs, but one is reminded of the shrewd remark of Sutro[2]: "If sea air alone can cure, your patients will not require foreign ingredients to be incorporated into their economy."

No account, however brief, of these natural remedies can omit the dangers of their injudicious or excessive use. According to the consensus of opinion, these are as follows: First, chill; i.e., lowering the temperature of the body by the abstraction of heat by baths in persons whose metabolism is already depressed. Cold is well borne if the metabolism is exalted, as in fevers and Graves' disease; it is ill-borne by those whose temperatures are subnormal. It is true that with some chilly persons, a momentary application of cold may be beneficial and stimulate the formation of heat. Not the *degree*, but the *duration* of cold, even as measured in seconds, may turn the benefit into a danger. Authorities in hydrotherapy have fixed 65° F. as the lower limit of temperature for immersion baths.[3]

The sea temperature on the east coast in summer ranges from 61° to 68°; on the west coast it may be from 5° to 10° higher. The lowering of a body temperature which is already subnormal, by placing a delicate child in water 40° below blood-heat, may have serious effects. Momentary exposure may be permissible, but for chilly children initial treatment by heat is better practice, and by rousing the dormant metabolism, may re-educate the defective toleration of cold. Although persons with a subnormal temperature should therefore bathe with caution, the milder stimulation of sea air is usually wholly beneficial. It is known that in health prolonged sea baths cause slowing and enfeeblement of the pulse and lowering of body temperature. It is said that the pulse may be lowered thirty beats per minute in the shrimp fishermen off Brighton, and that the blood temperature of the flounder fishermen off the North of Ireland is sometimes lowered 2½°. I do not know whether any of our members at Folkestone or Dover have made similar observations on long distance swimmers.

The second danger of the marine health resort is the aggravation of acute or febrile disease or of congested states. This is true, not only of sea baths, but to a less extent of sea air, and depends no doubt on the influence of both in stimulating or activating metabolic processes. Under this heading may be placed the aggravation of congestive rheumatism and gout. Reports reach me from some marine resorts that rheumatism and gout are contra-indicated, and that exacerbations of these disorders are not uncommon; whilst at other resorts it is stated that rheumatic and gouty cases do well. That is a point upon which the experience of practitioners round the coast might be of much value, especially in view of the extended treatment of insured rheumatic patients. Active pulmonary tuberculosis, hyperpiesia, acute catarrhs are of course included in this category.

Thirdly, what has been called "marine fever." A transient disturbance, marked by sleeplessness and slight pyrexia, is often met with after bathing, and may in sensitive subjects be much more marked. It indicates, of course, an overdose. In this last category ought to be included digestive disturbance and anæmia. Dickenson used to say, *apropos* of the West Country, that "anæmia is a marked instance of

1 Houzel, *Archives of Medical Hydrology*, January, 1925, 14.
2 "Spas and Climatic Resorts," 2nd ed., 1865.
3 R. F. Fox, "Outlines of Medical Hydrology," 1924, 36.

marine cachexia." It remains true that dyspepsia and anæmia are occasionally attributable to sea air and baths, probably owing to the aggravation of congestive states.

It remains to ask by what means the good effects of sea bathing can be promoted and its ill-effects avoided, especially in view of the seaward movement of our time. The burden of leading must rest upon medical men, who are responsible in one way and another for the public health. I suggest that a society, such as our own, devoted to the study of waters and climates, can perform a valuable service. It would surely be possible to institute and then to co-ordinate the observations and investigations necessary to enable medical men everywhere to obtain for their patients, and for the public generally, the optimum advantage at the marine resorts. In the first place, the proper conditions for popular sea bathing, especially for children, could be clearly defined and made known. Certain guiding climatic readings, including sea temperatures, are much to be desired. And secondly, a more difficult task, as regards the remedial use of sea air and sea baths, a wide field of study could be explored with real advantage. Many lines of investigation suggest themselves. What is the action observed at our various marine resorts on metabolism, especially in children, and upon body weight; on blood formation and circulation; on body and skin temperature and on immunity? Again, what is the precise reaction produced in different cases by the various methods of physical treatment employed. On these and other questions careful observations would be of much value, if standard measurements and records were employed. And they would lead up to the ultimate object of our research, namely, to define with some exactness the various local climates and physical methods of treatment favourable to different types and phases of disease.

[The activities of the Medical Society of the *Côte d'Azur* and of the International Association of Thalassotherapy meeting this year in Roumania were described.]

DISCUSSION.

Dr. PERCY LEWIS (Folkestone).

The chief factor in marine climates is, of course, the sea, which acquires and gives up heat slowly. Therefore the oscillations of temperature during the day are insignificant, and the changes in the year mild. The freshness felt at the seaside is due to there being always a breeze from the land to the sea in the evening, and from the sea to the land in the morning. The prevailing south-west winds from the Atlantic are warm, moisture laden, and free from solid particles. Thus the air lets through a maximum of light. It also has a high salt content which has been shown to be beneficial. The presence of ozone has not been unequivocally proved. The most important factor in the benefit obtained at the seaside is the abundance of sunlight; and the aspect of a place determines the amount it receives. The south-eastern district from Margate to Southampton, including the Isle of Wight, has sunshine above the average of any other district. This is due partly to the pure air, but more to the sea which acts as a gigantic reflector. Visitors to the seaside spend more time in the sunlight and wear less clothing than usual. Thus they are apt to suffer as do patients taking regular heliopathic treatment too rapidly. Heliopathic treatment should be graduated. At seaside schools the chief effect noticed is improved resistance to infectious disease.

Relaxing places are those with the highest temperatures, with smallest daily range and the largest degree of relative humidity. Bracing places have a lower temperature, a larger daily range and less relative humidity. A marine climate acts on children by exercising their adaptation and resistance without excessive strain, and this keeps the body in good working condition. Professor Beneke, of Marburg, showed that heat was more rapidly lost by any warm body at the sea-shore than by a similar warm body at inland or mountain localities. Increased loss of heat has to be compensated by increased production, and consequently seaside resorts necessitate increased activity of the heat-producing organs. A seaside climate seems, therefore, to be ideal for the physical development of normal children, or for

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increasing adaptation and resistance in abnormal children. The same results will obtain in all cases, whether infantile or adult, in which resistance has been lowered by disease, age, or operation.

In choosing a marine climate for a particular case, it is important to estimate the patient's power of reaction. If this be judged low, a relaxing place will be chosen, if high, a bracing one. One must also consider the time of year; e.g., cases of renal disease, pulmonary tuberculosis, and chronic bronchitis do well enough in the summer months in the bracing eastern and south-eastern places, but should not be sent there in the remaining eight months of the year. The power of reaction in these cases being notoriously low, relaxing or sedative places such as Dorset, Devon and Cornwall, are more suitable in the colder months.

A seaside climate depends mainly on the nearness of the warm, moist Atlantic winds. Therefore the east coast where the prevailing winds are from the land and consequently dry, is generally more tonic, while the west coast with the prevailing winds from the sea is generally more sedative. The most bracing district in south Britain is the Isle of Thanet. Here the sea is narrower, and therefore the sea-winds are drier than elsewhere. From Thanet to Bournemouth the bracing properties gradually decrease and beyond this, most places are sedative.

Sea-bathing is an excellent pastime for those who are well, but too much of a strain for those with lowered resistance. Salt-water baths in a bathing establishment are in a different category. Sea bathing is of minor importance in marine resort treatment except in so far as it involves increased exposure to light.

Dr. W. G. WILLOUGHBY (Eastbourne).

In calculating the possible value of a stay at the seaside for purposes of health, a most important point is the climate. Climate depends on many factors which vary from place to place, but certain climatic conditions which may affect health are common to all seaside places.

Between inland and seaside places at the same latitude, in addition to the points already mentioned by other speakers, the seaside district has a slightly higher barometric pressure, and, as a rule, less rainfall.

I submit a chart showing the principal meteorological conditions for a year in Eastbourne. In choosing a climate, the medical man often has insufficient detail: averages are deceptive. For instance, it is necessary to consider the mean of the daily maximum temperature and the mean of the daily minimum, rather than the mean generally. The mean generally on our South Coast is about 50°: this might be the mean of 45 and 55, or the mean of 80 and 20. The warmth and fitness for invalids of two places of such respectively varying temperatures would be utterly different. The total rainfall is usually given. The number of rainy days is much more important. In one place in the country the annual rainfall in one year was 80 per cent. over the average, whereas the number of rainy days was only 15 per cent. over the average. It is necessary to obtain the conditions for the time of year when the change is to be made, and if detailed charts were published medical practitioners would have much more helpful data.

As to how the different conditions at the seaside affect the health of the patient, experiments are wanting and experience has to be relied upon. Professor Beneke found that the seaside air produced an acceleration of the nutritive changes in the nitrogen-containing tissues of the human body and that body heat was lost and regained more rapidly, but more continuously, at the seaside than in mountain districts. It is necessary to remember, however, that Beneke took extreme altitudes.

It is in the later stages of convalescence probably that the seaside most favourably affects the patient. Experience has shown the value of some of the factors of climate which I have mentioned, particularly sunshine—the freedom from dust in the air is also important—owing to the proximity of the sea there is an increase in light. Even the slight increase of oxygen also leads to quicker tissue change. The equality of temperature due to the proximity of the sea is satisfying to invalids, and this equality of temperature enables weakly persons to be longer out of doors.

I consider it very important that children should not live in too relaxing a climate.

As regards the humidity of seaside resorts, too much is made of this, and of the effect of trees unless the amount of trees is such that they become a forest. I have compared the

relative humidity over twenty-five years in five-year periods of Brighton, London, Eastbourne, and Southport. The humidity of Brighton in every one of the five-year periods was less than that of London. The humidity of Eastbourne was slightly more than London in four of the five-year periods. The humidity of Southport was less than that of London in three of the five-year periods, but the whole of the difference amounted to very little—the extremes were 80 per cent. and 86, but humidity was almost always about 82 per cent.

Apart from illness, in the general lives of healthy children, in this country the difference in climate in various districts is so comparatively small that the social conditions more than counterbalance any climatic effect, that is to say, that if good food, good clothing, proper housing are provided, the place where the child lives does not seem to have much practical effect on height, weight, and physical development.

An important feature in marine resorts, besides climate, is the provision of other amenities, for the mind, as we know, has a very great effect on the body. Sea-bathing for instance is a very great attraction for children, but it has to be carried on with considerable care, especially after illness. The people who take hot sea-water baths report how much they seem to benefit from them, and what a feeling of well-being they give.

There might with advantage be much more recording of effect of "change to the seaside" on patients with various affections, and it would be very useful indeed to get a collection of reports of practitioners as to the effect of the change on their patients.

Dr. COLLIS HALLOWES (Torquay).

Each marine resort has its own advantages and disadvantages, and the individual's response to the climatic conditions at the selected resort is the deciding factor. To one person the resort is stimulating, to another sedative.

Growth and development during childhood are favourably influenced by a marine climate; the resisting powers of invalids are raised by an open-air life, sunshine and ultra-violet rays. Sea breezes have many advantages.

The climate of Torquay, characterized by its equability, low mean daily range of temperature, moderate rainfall, low humidity, high average of sunshine with high percentage of ultra-violet rays, dry soil and excellent shelter from the north and east, is very suitable for children, elderly invalids and the aged, and for convalescence after acute illness or operation.

For children the prophylactic use of such a climate is of importance. Children with evidence of deficient thyroid secretion are much benefited. It is interesting to speculate as to whether their improvement may in part be due to the increased percentage of iodine in the air. Subacute bronchitis and chronic bronchial affections are favourably influenced; and pulmonary sequelæ following acute illness will probably be prevented by change to such a suitable marine climate. Many sufferers pass through the winter there without an acute or subacute bronchial attack. Some asthmatics, especially those of the bronchial type, do well. People live long at Torquay, elderly invalids and the aged are not subjected to the strain of a more bracing climate. Life is thus made more tolerable, and pulmonary affections may be prevented or delayed.

Chronic nephritis and cardiovascular lesions tend to become more quiescent; Raynaud's disease and "chillblain circulation" benefit from the equable conditions.

It is often stated that the rheumatic group of diseases is not beneficially influenced by a marine climate. This is certainly not true of all marine health resorts. Cases of fibrositis, neuritis, arthritis and peri-arthritis, do extremely well at Torquay, which is fortunate in possessing a complete and modern establishment where all forms of hydrotherapy and many physio-therapeutic treatments can be given, and in my experience the results are good. In winter, treatment can be continued without the risks associated with more bracing resorts.

Invalids should bathe in the sea or out of doors only on medical advice, but generally they will derive benefit from hot sea-water baths and tepid sea-water swimming baths, both in winter and summer.

Post-operative breakdowns are not infrequent, especially in middle-aged women and men of the sedentary type; these could probably be prevented in many cases by insistence on prolonged rest and change at a health resort under suitable conditions and medical observation. The strain to the nervous system incidental to a surgical operation, often seems to receive only secondary consideration, and return to work is permitted at too early a date. Again, operative procedures are often undertaken when the resisting powers are much

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below normal—e.g., the removal of a focal infection in the rheumatic—and little benefit is frequently derived from them. I feel confident that if the resisting powers were stimulated previous to operation, by a sufficient period of climatic treatment at a health resort, with a high sunshine average, results would be infinitely improved.

Dr. W. S. CAMPBELL (Sidmouth).

I have practised on the coast for many years and have had the opportunity, on the Northumbrian and Devon coasts, of observing the effect of the seaside upon health.

At Whitley Bay in Northumberland the climate is cold, dry and windy, the country is flat and devoid of trees. At Sidmouth the climate is warmer, more moist, less windy, the country is hilly and well wooded and there is a much greater amount of winter sunshine.

The therapeutic effects observed shortly after arrival at both these places are that patients sleep and eat better, and after some days become capable of more exertion, thus showing that the functions of the body are being carried on more efficiently, and in consequence disease is more vigorously combated and convalescence is hastened. Children react to this stimulus more quickly than do adults, and I found that children were equally benefited in the north-east and in the south-west provided that they were sent to the seaside at the appropriate season. Whitley Bay is purely a summer resort, while Sidmouth, on account of its greater winter sunshine, is an all-the-year-round one.

It is therefore apparent that the two factors involved in the success of seaside treatment are the sea and the sun, and that climate has little to do with the matter. I have discarded the possibility of iodine vapour being a factor. It is stated that goitre does not occur in places near the seaboard, but in a village near Sidmouth goitre is very common. This yields quickly to treatment by iodine, so it seems unlikely that the iodine from the sea travels far or has much potency.

We have now to consider the effect of light falling upon the large reflecting surface of the sea, and I believe this to be reason for the success of marine health resorts, and that this success is in direct ratio to the amount of reflected light, especially from the rays of short wave-length.

It is estimated by Dr. Brown and Dr. Tisdall that the amount of ultra-violet rays from "skyshine," that is, the reflection of rays from the clouds and sky, is one-half to two-thirds of that of the direct rays of the sun. I do not know the amount of ultra-violet rays reflected from the sea, but this "seashine" must be a good deal more potent than "skyshine."

This is evident when we consider the question of tanning of the skin, which is a fairly delicate method of estimating the amount of ultra-violet rays. Tanning will occur in any townsman when he is exposed to the direct rays of the sun, whether he be at the seaside or in the country, but when there are no direct sun's rays tanning takes place only at the seaside, on the sea, or on a snow-clad mountain.

Dr. Leonard Hill has said that "biological evidence shows that the region about 3,000 has the principal action on the skin, causing sunburn and in activating ergosterol and in producing antirachitic vitamin," so that there must be a considerably greater amount of these rays, produced by seashine and skyshine combined, at the seaside than is produced by skyshine alone in the inland country.

Reflection increases the intensity of diffused light, and the shorter wave-length rays are more easily diffused than the longer wave-length. Therefore we find that the diffused light coming from the sea possesses a larger proportion of ultra-violet rays than inland light.

Watery vapour causes diffusion of light and therefore the intensity of the diffused light is greater at low levels than at high and at the seaside greater than in the dryer parts, but at the same time we must remember that watery vapour holds up some of the ultra-violet rays. The uneven surface of the sea aids the diffusion by reflecting in various directions and probably increases the intensity. In addition to this there is no smoke pollution to hold up the ultra-violet rays.

So that at the seaside we get the effect of heliotherapy without the necessity of actual sunshine, because "seashine" is continually producing ultra-violet rays.

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Mr. W. G. SUTCLIFFE (Margate).

Margate has been known as a health resort since the end of eighteenth century. The Royal Sea Bathing Hospital founded in 1791 was the first institution of its kind in the country, it now possesses 300 beds and 100 of these are reserved for children. Other institutions are the Princess Mary Hospital for Children, under the care of the Metropolitan Asylums Board (200 beds) and the Victoria Home for Invalid Children (60 beds). These are set apart for cases of surgical tuberculosis, and 30 years' work in Margate has given one plenty of opportunities for observing that disease. Long before the alpine methods of heliotherapy had become fashionable and advertised, the climate of Margate with its sunshine, exposed frontage, bracing winds, and dry soil, had been a factor in the treatment of cases sent from all parts of the kingdom.

It was found that those that were exposed during the whole day on promenades, sands or balconies, rapidly improved in general health, and with that improvement amelioration in the local conditions could be looked for with abiding certainty. There is no season of the year in which children, and for the matter of that, adults, cannot be exposed to the air in Margate. The winter, is as a rule, dry and bracing, the prevailing winds are nearly all sea breezes either coming from the north-east or up the channel from the south-west; and three consecutive days without sunshine are extremely rare.

The recent work of Hill and Campbell on the increase of basal metabolism, goes far to explain the beneficial effects accepted empirically for many years, and we are now preparing, by the Leonard Hill acetone methylene-blue indicator, a detailed system of records of ultra-violet ray activity which cannot fail to add materially to our knowledge. Increases in metabolic activity, judiciously controlled, are undoubtedly a prime factor in promoting the tissue repair that is needed in surgical tuberculosis. That they require control has been observed again and again, especially in treatment of tuberculous glands which has long been one of the special features of Margate. When comparatively early cases arrive with enlarged glands in that condition of unstable equilibrium in which resolution has not begun and caseation not taken place and the patients are allowed to play on the sands, paddle and bathe, the increase of basal metabolism is the cause of too great an auto-intoxication, and softening, caseation and abscess will take place. We have therefore always insisted on a period of practically complete rest until the condition in the glands subsides. By far the greater number, if treated in time, will disappear without operative treatment, or assistance from the various forms of artificial heliotherapy with which we are now abundantly provided. In Margate, the treatment of tuberculous joints has always been of the conservative type now adopted everywhere, and the more modern system of more or less complete exposure of the whole body has undoubtedly added to the number of good results.

[Dr. Sutcliffe showed a number of photographs taken by Dr. West at the Princess Mary Hospital, illustrating the treatment by exposure as carried out in Margate.]

Dr. OCTAVIA WILBERFORCE (Brighton).

My experience has been that children under the rather special conditions of Brighton pick up quickly after the usual childish complaints. It is difficult to estimate to what extent our daily average of sunshine hours (4·81) is responsible for this fact.

There are a considerable number of cervical adenitis cases, some obviously due to septic tonsils, others to tuberculosis. Severe cases of acute rheumatism and rickets seem to come from adjacent and often rural districts rather than from Brighton itself. Skin troubles are notoriously capricious. A child, whose home is in the Midlands, was at death's door from prurigo gravis. She was restored to comparatively normal conditions by Brighton treatment. Since then, eighteen months ago, her condition has to be periodically ameliorated by visits to Brighton. Unexpectedly enough, the West Coast of Scotland is found to serve equally well.

Pulmonary tuberculosis and advanced bronchial trouble both do badly. The place is too bracing and the winds may sometimes be too keen.

There are three distinct climates in Brighton:—

(1) Kemp Town, the east end, is higher, windswept, rather bleak, and very stimulating.

(2) Hove, the west end, is considerably warmer, lower, more sheltered. A part of Hove, however, is on clay soil.

(3) The northern part of the town combines the bracing sea characteristics with the more balmy air coming off the South Downs.

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Two cases illustrate this point:—

A boy aged 5½ from Herefordshire came to me first in 1924. He had been a healthy baby until the age of a year and ten months, when, it was said, he had influenza. He had had bronchial asthmatic attacks every few weeks since. I found that he had a protein sensitization, vomiting after meat, milk, and eggs. An acute attack always followed a visit to the barber. Peptone injections and ultra-violet radiation treatment improved his condition but until March, 1927, he certainly was not cured. His brother, aged 3¼, was so ill (suspected tuberculous peritonitis) that a local Herefordshire practitioner in desperation advised Brighton. I learned that the child had had urticaria ever since he was six weeks old.

Both boys had broncho-pneumonia in December, 1924. Frequent attacks of urticaria accompanied by vomiting and pyrexia were, if caught early, often curtailed by adrenalin, but since March, 1927, i.e., a year, neither boy has been ill.

I attribute these dramatic cures to a change of residence. The family moved to an older house scarcely half a mile further north, more open, with air from the South Downs, and on gravel soil.

Children who live at Brighton often become sleepless and nervous unless they go away for a change from time to time.

Comparing fifty Brighton children of various classes with fifty convalescents from poor London homes, I find the average weekly gain of the residents is from 9 to 16 oz. Those only were weighed whose progress did not satisfy me. The fifty visiting children at an institution were all weighed on arrival and departure. Their average weekly gain was 8 oz.

In these days of longevity, and of efficient longevity, out of fifty patients, in the last five years, whose ages were between 70 and 87 (average age 75), forty-four have survived. Their general condition has altered surprisingly little: eight with arthritis have remained about the same, five with hyperpiesia (a systolic blood-pressure over 200) have not progressed more than to reduce their activities slightly. One with threatened gangrene of her right little toe, aged just 80, grudgingly consents to having her breakfast in bed but otherwise remains remarkably the same. Three years ago her systolic blood-pressure was 270, now it is 220 systolic, 100 diastolic.

Often the elderly bronchitics do better than the bronchitic children.

As regards convalescents, there are plenty of diversions for all ages and the bracing air puts an edge on appetite and induces sleep.

One woman, after three operations for breast abscesses in London, recovered completely, and gained 14 lb. in seven weeks.

A man aged 50, with arterio-sclerotic gangrene of the left heel and little toe, came to Brighton in November, 1926. For over four months morphia had been necessary to ensure any sleep. A periarterial sympathectomy in October disclosed arteries too sclerotic to derive much benefit. His emotional self-control had entirely broken down. He would weep like a child with the pain and in spite of heroin it was nothing for the nurse to be called up four or five times in a night. After eight weeks he had regained his former equipoise and was sleeping reasonably well with no other sedative than sedobrol. Even the local condition was definitely better, though never completely healed.

Dr. W. B. BARCLAY (Weymouth).

Both the myrtles flourish and blossom at Weymouth, an evidence of the mildness of the climate. As a sea-bathing station the place has special advantages; position on a promontory: soft firm sands, with a gradual declivity, and a well-sheltered bay. Experience has shown that tuberculous patients do very well in many cases, as do those with myocardial weakness, psychasthenia and neurasthenia. Weymouth occupies a middle position between the tonic influences of the east and the sedative influences of the west. The health of children, according to the Board of Education's returns, is exceptionally good.

Dr. FOX (in reply),

commented upon three important features of the marine health resort which had been brought out in the discussion. Dr. Willoughby, from his long experience, had shown that the seaside provided an environment of unique *interest*, which had a psychological influence in chronic disease. Dr. Hallowes showed what sea-air could do for delicate persons *before operation*, a fact of which few surgeons were aware. Dr. Bruce[1] had used the word *arrest*, in regard to tuberculous cases at Hastings. The word often described the effect of the marine resort not only on tuberculosis but on many disease processes. Dr. Sutcliffe's demonstrations of the results at Margate were convincing and deserved to be much more widely known.

[1] Report not received.—ED.

Clinical Section.

President—Sir HERBERT WATERHOUSE, F.R.C.S.

[*March* 9, 1928.]

CASES.

Subluxation of the Head of the Fibula in a Boy, aged 10.

By CECIL P. G. WAKELEY, F.R.C.S.

J. M., AGED 10, fell down in January, 1928, while playing football in the playground and hurt his right knee. The knee became slightly swollen, and the boy could walk, but running was painful. When seen two weeks after the injury the head of the fibula was subluxated and could be moved in every direction.

X-ray examination demonstrated that there was a definite interval between the head of the fibula and the outer tuberosity of the tibia.

The boy is having massage to the muscles in the region of the knee, while the head of the fibula is kept in place by means of a pad and bandage.

I think no operation is indicated.

Sir HERBERT WATERHOUSE (President) agreed that it should be left alone; he had known of a similar injury from which the patient had recovered without operation.

Compound Follicular Odontome of the Upper Jaw in a Woman, aged 42.

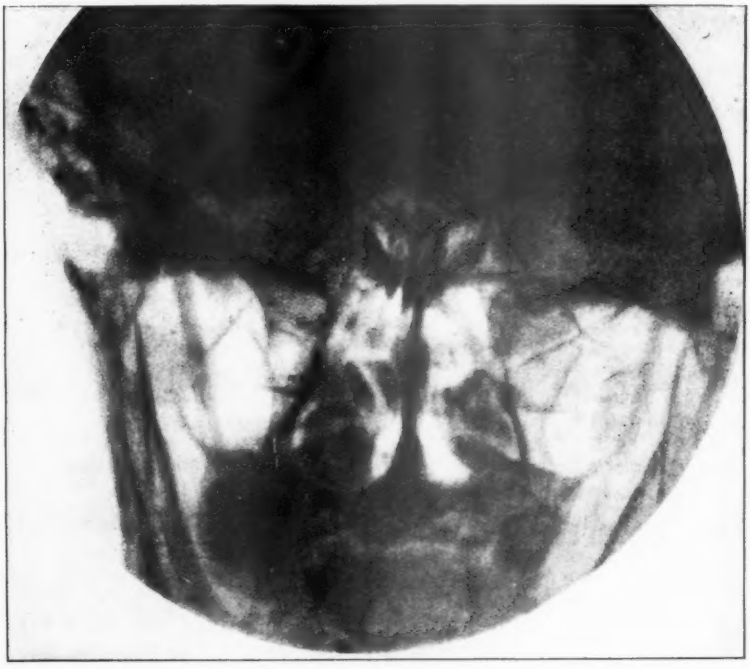
By CECIL P. G. WAKELEY, F.R.C.S.

S. W., NOW aged 42, noticed at the age of 35, that the right side of her upper jaw was enlarged slightly. At this time she had some teeth removed from the upper jaw, and a few years later she had all her remaining teeth extracted and double dentures fitted. The upper denture required remaking at the end of 1927, and her dentist, Dr. Moodie, of New Cross, sent her up to King's College Hospital in January, 1928. There is definite enlargement and expansion of the alveolus on the right side of the upper jaw. This is very hard and is definitely incorporated in the bone.

Skiagram shows a definite compound follicular odontome containing several teeth.

78 Wakeley: *Compound Follicular Odontome of the Upper Jaw*

The dentist is not sure that there was a complete set of teeth in the upper jaw, so there is some doubt about the missing teeth. I saw the case at the end of 1927, and we had a cast made of it (skiagram shown). There is a definite tumour, and there are more opaque spots. It is mainly because of the X-ray appearances that I have called it a compound follicular odontome, but it might be an epithelial odontome; it is harder than the former usually is. Has the dental plate irritated



Compound follicular odontome.

the lining of the cyst and so caused bone to be thrown down? In the cast of the upper jaw the bad side is enormously dilated, more on the outer side than on the inner. Operation is necessary, and I think it will be difficult to get the tumour away.[1]

Sir HERBERT WATERHOUSE (President) thought the tumour was an odontome. Fluid was absent; there were too many black spots (four) for them to be malformed teeth. No doubt an operation was necessary and it promised to be difficult. He had not seen a case exactly like this with so long a history.

1 *Postscript.*—Operation was performed March 15, 1928. The whole bony mass was removed quite easily. It proved to be a small compound follicular odontome.

Bilateral Clicking Jaw in a Woman, aged 39.

By CECIL P. G. WAKELEY, F.R.C.S.

MRS. M. N., aged 39, first noticed about two years ago that the right temporo-mandibular joint clicked during mastication. This clicking has become more pronounced of late, and the left joint now also clicks. On the right side the intra-articular fibro-cartilage can be felt to be subluxated at every click of the jaw.

I have only operated upon two such patients, and both cases were very bad, practically having subluxation of the inter-articular fibro-cartilage and marked osteo-arthritis of the joint, which are not present in this case. I have removed the cartilage and put in sterilized vulcanite, stitching it in position, and the cases have done well. I think this case should be left alone, but when people persistently come up and complain of the noise, we should excise the cartilaginous portion of the condyle.

Sir HERBERT WATERHOUSE (President) said that in many such cases he had refused to operate. He had made an exception when the wife of the patient threatened divorce if the nuisance was not stopped; he had never previously seen such a case in a man.

A Large Dental Cyst.

By CECIL P. G. WAKELEY, F.R.C.S.

A. E., MALE, aged 37, first noticed a small swelling in the left side of his face in May, 1927; this has been growing larger and is now the size of a golf ball.

There is a rounded tumour 1½ in. by 1 in. just below the left malar bone. It is unattached to the skin, but is firmly attached to the bone beneath. Definite fluctuation can be made out. The swelling appears to originate from the lateral wall of the left superior maxilla.

The swelling was first painless, but is becoming more sensitive; deep palpation is painful.

The patient has three or four septic roots in the left upper jaw and is wearing a denture over them.

The left antrum is opaque to transillumination and to X-rays, which latter show the walls of the maxillary sinus as intact; hence it is very unlikely that the swelling originated from that part of the bone.

Postscript.—Operation was performed March 15, 1928. The septic stumps were removed, and during the extraction of the last, the swelling discharged through the empty socket. It proved to be a dental cyst.

An Innocent Intrathoracic Tumour.

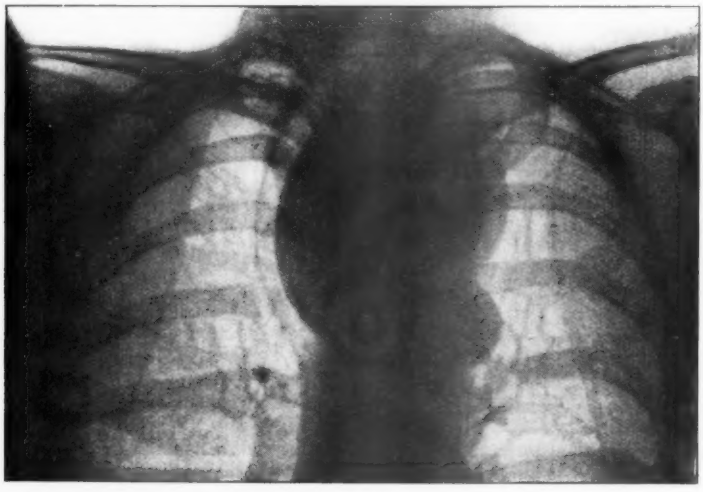
By F. G. CHANDLER, M.D.

MRS. W., aged 56. For eighteen months has complained of violent pains across the chest in the mid-sternal zone. Has lost much weight since July, 1927. Has had stridor for many years. Twelve months ago an X-ray examination was made and aneurysm diagnosed.

Past History.—Had a cough in 1915. Then attended hospital for twelve months. No X-ray examination. Patient's doctor states that she has had symptoms of exophthalmic goitre for some years, but no obvious enlargement of the thyroid. In 1923 began to have some pain in the chest.

80 Chandler : *Two Cases of Innocent Intrathoracic Tumour*

Present Condition.—No cough, or expectoration, or blood. Stridor. No other pressure symptoms except some enlargement of veins in the neck when eating. No goitre. There is dullness over the manubrium sterni, and on each side of it ; below this dullness becomes continuous with the heart dullness. No pulsation. Aortic second sound normal. No murmur. Pupils and pulses equal. No glandular



enlargement. Wassermann reaction negative. Screen examination reveals a large tumour in the upper part of the thorax, displacing the trachea to the right. The trachea can be seen very clearly passing round the right edge of the tumour. No pulsation. Skiagram confirms this appearance. A barium paste examination shows the œsophagus to lie behind the tumour, but to be displaced slightly to the right.

A Specimen of Innocent Intrathoracic Tumour (? Fibroma) discovered at the Post-mortem Examination of a Woman killed in an Accident.

By F. G. CHANDLER, M.D.

Pathological Summary.—Fractured pelvis. Tumour in posterior mediastinum. Left lung : Adherent at apex ; weight 220 grm. ; scarring at apex. Right lung : 200 grm. ; nothing abnormal. Ribs : Sixth right rib fractured. In the thorax on the right side of the fourth, fifth and sixth vertebral bodies was a large tumour, 5 in. in its long diameter, by 3 in. by 2½ in., lying external to the pleura. On section the tumour presents yellowish patches, hyaline areas, cystic spaces and hæmorrhages. *Microscopical appearance* suggests fibroma.

From inquiries from relations it was discovered that no symptoms were complained of during life.

Discussion.—Dr. L. S. T. BURRELL said he thought at first that in the first case the tumour was connected with the thyroid. The tumour in the second case he regarded as a fibroma. He had not heard of such a condition being found accidentally before.

Mr. A. TUDOR EDWARDS thought the first case suggested substernal or mediastinal goitre. The thyroid swelling could be felt, but when the patient swallowed, a tumour was felt coming into the neck. He (the speaker) had had five cases of intrathoracic goitre in the past year and they all gave a well-marked outline, as in this case. A lateral photograph showed them anteriorly.

In such a case the treatment depended on several factors. There was stridor and breathlessness, and should pain increase, something must be done. This type of goitre, which was broader below, could not usually be removed through the upper aperture of the thorax, as described by Sir James Berry for substernal goitres. In three cases he (the speaker) had been obliged to split the sternum, not a difficult operation nor one causing much shock. Usually these tumours were well encapsuled, and though large veins led into the capsule they presented no great difficulty. One did not enter into the true mediastinum, but kept within the capsule.

The record of Dr. Chandler's second case was interesting to him personally, because he had recently had one of a similar type in a woman who had had acute pain round the chest. After full investigation the chest was opened and the tumour removed; the operation was not difficult. The tumour was described as a fibro-lipo-sarcoma, consisting of sarcomatous cells with embryonic fatty tissue. That was two years ago; he had seen the patient recently, and there was no evidence of recurrence from a skiagram taken a fortnight ago. The section of the tumour now shown consisted of fibrous tissue, but if a portion from another part was examined microscopically, further light might be thrown on its nature.

Mr. T. P. DUNHILL said he felt sure that the tumour in Dr. Chandler's first case was an intrathoracic goitre, both from the appearances of the tumour itself and from the history. The tumour had a well-defined border. It was oval in shape, as is frequently the case with an intrathoracic adenoma of the thyroid. He had operated upon two cases of dermoid, and had seen others. They were almost always spherical, not elongated as this was. He had skiagrams of an identical case in which the tumour had been proved to be thyroidal in origin. This patient had suffered from goitre for many years. The tumour had disappeared from the neck synchronously with the commencement of the present symptoms. Now no thyroid could be detected in the neck.

He had had one case in which the thyroid tumour had extended to the level of the origin of the seventh rib.

Regarding treatment: these adenomata tended to become toxic in the late middle period of life. This patient was rather better than she had been in some respects. Her cough and expectoration had disappeared, but she was suffering from pain. If the pain was sufficient for her to wish an operation so that she could the better do her work and enjoy life, he would not hesitate to do one. He believed that she could be operated upon safely and that it was a matter of balancing her present discomforts against the troubles incidental to operation. He thought that the symptoms would ultimately tend to increase. When a woman had had a large goitre for the greater part of her life, and at about the period of the menopause it began to give trouble, it was common for the heart to be affected more than the nervous system. He would prefer to deal with the condition while the patient was still strong. In a number of similar cases he had removed the tumour by splitting the upper part of the sternum and cutting outwards into the appropriate intercostal space. This permitted efficient access to the superior mediastinum. It was the experience of most goitre clinics that in 1·6 to 2 per cent. of cases of this type of goitre, malignancy supervened. His own experience exactly corresponded with this. In the case in which the tumour had descended to the seventh rib, the patient had been operated upon in a London Hospital ten years before he saw her, and the tumour in the neck removed. It was reported to be malignant. When she went on for years without recurrence it was thought a mistake in diagnosis had been made, and he thought so too. On microscopical examination of the tumour removed, it was found to be a malignant adenoma. The patient had died subsequently from local recurrence.

With reference to the second case, he (Mr. Dunhill) had operated upon one almost identical. The patient had suffered from recurrent attacks of bronchitis, and, while sweating occurred normally on the left side, it was absent on the right side of the face and right arm. These attacks became more frequent and severe. The man was a patient of Dr. J. H. Drysdale, who had worked out the condition and had had an X-ray examination made. This revealed a large tumour, circular in outline, situated in the upper part of the right thorax. He (the speaker) had removed this tumour, access being obtained by means of a sternum-splitting operation reaching the third interspace. The patient was operated upon in 1921. His recovery was uneventful and he had lived an active life since. The tumour had been attached to the right side of a dorsal vertebra, probably the third or fourth. Microscopical examination showed it to be a fibroma. This case was reported in the *British Journal of Surgery*, 1922, x, 37.

Dr. CHANDLER (in reply) agreed that in his first case the tumour was probably a substernal adenomatous goitre. When aneurysm was diagnosed the patient had been treated with iodide of potassium, and she had been much better since, a fact which perhaps tended to confirm the idea of thyroid tumour.

He agreed that sections from the specimen in the second case should be cut in different places for histological examination as many innocent intrathoracic tumours had a most peculiar structure. For example, Jacobaeus had described the tumours in two of his cases as fibroma-myomata and xantho-sarcoma, and Mr. Tudor Edwards; in his paper in the *British Journal of Surgery*, 1927, had described one tumour as a fibro-lipo-sarcoma. In Wilson Fox's "Treatise on Diseases of the Lungs and Pleura," in addition to fibromata, steatomata, lipomata-sarcomatodes and sarcoma-lipomatodes were described.

Mediastinal Tumour.

By L. S. T. BURRELL, M.D.

D. P., MALE, aged 56. Gas worker. Shortness of breath began gradually in 1920. Cough and sputum developed about the same time, being worse in winter. Some five years ago he had an operation for removal of cancerous warts. He still has some warts on the hands and face. In March, 1927, X-ray examination of the chest showed a mediastinal tumour. Since then he has had treatment by radium. In January, an ulcer formed on the back and was excised for microscopical examination as it appeared to be connected with the radium applications. Skiagram taken March, 1928, shows the present condition.

High Blood-pressure, with Thrombosis of a Retinal Arteriole.

By F. PARKES WEBER, M.D.

THE patient, Mrs. F. D., aged 44, is a rather fat, florid English woman, who has, she thinks, mostly enjoyed good health. She says that at the age of 30 she had rheumatism of some kind. Four years ago she noticed a sudden "change" in her sight but never saw a doctor about it. Her brachial blood-pressure is very high: 250 mm. Hg systolic, and 140 mm. Hg diastolic (February 21, 1928). There is some hypertrophy of the heart to the left, but no cardiac murmur. The urine contains a trace of albumin and a few granular tube casts (no sugar). She has not to get up at night to pass urine. The lower part of the legs above the shoes shows a slight degree of the erythematous condition, now termed (on the Continent) "erythro-cyanosis frigida crurum puellarum (feminarum)," a condition much more frequently seen in girls and young women.[1] The Wassermann reaction is negative.

1 F. Parkes Weber, "Note on a kind of Chronic Erythema of the Legs in Girls and Young Women," *Brit. Journ. Derm. and Syph.*, London, 1925, xxxvii, p. 259.

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Dr. C. Markus (February 20, 1928), kindly gave me the following report on the eyes: The lower temporal branch of the central artery of the retina of the right eye is converted into a white strand from the edge of the optic disc for about the length of a disc-diameter. In the sector supplied by this arteriole are two bigger and numerous fine retinal hæmorrhages (? hæmorrhagic infarction). Corresponding to the occlusion of the retinal arteriole there is loss of the visual field in the whole of the upper inner quadrant. The visual acuity (that is to say, the central vision) is normal. There is nothing abnormal with regard to the left eye.

Remarks.—Geoffrey Evans thinks that the anatomical basis of arterial hyperpiesia is what he describes as diffuse hyperplastic sclerosis[1] of the arterioles, for which Aschoff has used the word "arteriolosclerosis," to distinguish it from the coarser arteriosclerosis of the bigger vessels. It is interesting in the present case to find such involvement of one of the arterioles in the retina. Affected arterioles are doubtless particularly prone to angiospastic attacks, during which local thrombosis may occasionally take place, and this almost certainly occurs in regard to the retina.[2] But it is not only arterioles that may be affected with angiospasm. I have witnessed the temporary apparently angiospastic disappearance of one radial pulse in an arteriosclerotic subject, and temporary cerebral "strokes" have been ascribed to similar angiospasm of cerebral arteries.[3]

With regard to high degrees of arterial hyperpiesia (hypertension) in relatively young subjects, I believe, in accordance with the work of Starling and Anrep, and Bordley and Baker, that the hyperpiesia is less the expression of generalized arteriolosclerosis than of an arteriolosclerotic disturbance in the blood-supply of the medulla oblongata. In a discussion on the subject in January, 1926, I expressed my opinion[4] as follows: "Professor E. H. Starling has admirably explained and experimentally illustrated[5] that the normal brain needs a well-maintained blood-pressure and that, if there is danger of the blood-supply in the vasomotor centre and brain becoming insufficient, the blood-pressure of the whole body will be raised in order to maintain the blood-pressure in the brain at a sufficiently high level. When active participators in the serious business or strenuous pleasures of life reach a certain age, say, about 50 years, some of them begin to react to the stress of life by increasing blood-pressure. This, I take it, is in many cases because the cerebral circulation has become relatively defective, owing to changes in the arterioles (and capillaries?), or because the nervous centres themselves have become blunted in some way . . . and demand a higher blood-pressure to enable them to perform the work . . . demanded of them." Bordley and Baker[6] have indeed examined the arterioles of the medulla oblongata in a series of cases of arterial hyperpiesia (hypertension) and have come to the conclusion that Starling and Anrep's work does really explain the pathogenesis of hyperpiesia, and they have themselves apparently demonstrated that at least most cases are due to arteriolosclerosis of the medulla oblongata.[7]

1 G. Evans, "A Contribution to the Study of Arteriosclerosis," *Quart. Journ. Med.*, Oxford, 1921, xiv, pp. 215-282; and *idem*, "Goulstonian Lectures on the Nature of Arteriosclerosis," London, 1923 (Lecture iii).

2 *Cf.* F. Parkes Weber and R. Gruber, "Recurrent Temporary Amblyopia of Angiospastic Origin and the Association of Retinal Angiospasm with Vasomotor Neuroses," *International Clinics*, Philadelphia, 18th series, 1908, ii, pp. 111-124.

3 W. Russell, *Brit. Med. Journ.*, 1909 (ii), p. 1109; *Lancet*, London, 1912 (ii), p. 1349; *Brit. Med. Journ.*, 1914 (i), p. 1057; also J. G. Sharp, *Lancet*, London, 1915 (ii), p. 863.

4 F. Parkes Weber, Discussion, *Proc. Roy. Soc. Med.*, General Reports, 1926, xix, p. 33.

5 Starling, *Brit. Med. Journ.*, 1925 (ii), pp. 1163-1165; see also Anrep and Starling, *Proc. Roy. Soc.*, London, 1925, xcvii, B, p. 463.

6 J. Bordley and B. M. Baker, "A Consideration of Arteriosclerosis of the Cerebral Vessels and the Pathogenesis of Hypertension," *Bull. Johns Hopkins Hospital*, Baltimore, 1926, xxxix, pp. 229-264.

7 According to H. Kahler ("Die Blutdrucksteigerung," *Ergebn. d. inn. Med. u. Kinderheilk.*, Berlin, 1924, xxv, pp. 265-413), in high blood-pressure of cerebral origin, lumbar puncture ought to lower the blood-pressure, at least in some cases.

84 Parkes Weber: *Erythrocyanosis Frigida Crurum Puellarum*

In conclusion I would suggest the following:—

(1) In the present case and similar cases the permanent obstruction of a retinal arteriole is due to thrombosis following local angiospasm, probably in connexion with local arteriolosclerosis. Embolism is unlikely as a cause in such cases, especially when no obvious source for embolism can be discovered. Similar obstruction of arterioles in other parts of the body in arteriolosclerotic patients (with or without extremely high blood-pressure) often doubtless gives rise to no definite symptoms, but I suggest that some peculiar spots of trophic disturbance in the skin may be due to local occlusion of arterioles (possibly with local sympathetic nerve involvement), for instance, a locally recurring solitary bulla.

(2) In similar cases of arterial hyperpiesia, especially in relatively young individuals, the occurrence of permanent occlusion of an arteriole in the medulla oblongata (like that of the retinal arteriole in the present case) would be a sufficient theoretical explanation to account for the permanent hyperpiesia. Temporary paroxysms of arterial hyperpiesia (possibly precipitating an attack of cerebral hæmorrhage) might be theoretically explained on the supposition of a local temporary arteriolospastic condition in the medulla oblongata (in a position not causing cardiac syncope or disorder of breathing) in connexion with arteriolosclerosis.

Discussion.—Dr. HALLS DALLY said that a case of hyperpiesia which he had shown two years previously,[1] might, although occurring in a woman, aged 62, i.e., older than Dr. Parkes Weber's patient, be held to support the view that most cases of hyperpiesia depend upon arteriolosclerosis of the medulla oblongata. During 1925 arterial pressure in his (Dr. Halls Dally's) case had reached the extremely high figures of 320/160, with no detectable evidence of sclerosis of the vessels elsewhere than in the fundi, with no hæmorrhages, and visual acuity unimpaired. Fairly good health had been maintained up to the present, the pressures now varying little from 250/135.

Dr. EVAN BEDFORD said that we should be cautious before concluding that, because certain arterial changes were found in the medulla, they necessarily caused hypertension. They were certainly associated with hypertension in some cases, but in the paper quoted by Dr. Parkes Weber similar arterial changes had been found elsewhere than in the medulla.

Dr. PARKES WEBER, in reply to Dr. Bedford, said that arteriolosclerosis was a more or less generalized condition, but might involve the medulla oblongata before the kidneys were much affected. Moreover, extensive arteriolosclerosis of the kidneys was not invariably associated with very high blood-pressure.

Erythrocyanosis Frigida Crurum Puellarum.

By F. PARKES WEBER, M.D.

THE erythematous or chilblainy condition of the legs in girls and young women which I discussed in 1925, may be explained as representing an abnormal reaction—that is to say, a reaction occurring only in abnormally predisposed or idiosyncratic subjects towards the local application of cold, the local effects being favoured by the scanty clothing of the affected parts, in accordance with prevalent fashion.[2] The condition, which varies very much in degree (colour, amount of swelling, etc.), in

1 *Proc. Roy. Soc. Med.*, 1926, xix, No. 4 (Clinical Section) p. 13.

2 F. Parkes Weber, "Note on a kind of Chronic Erythema of the Legs in Girls and Young Women," *Brit. Journ. Derm. and Syph.*, London, 1925, xxxvii, pp. 259-262; *idem*, "Two Diseases due to Fashion in Clothing," *Brit. Med. Journ.*, 1925 (i), p. 260; *idem*, "Two Cases of Chilblainy Condition of the Legs," *Proc. Roy Soc. Med.* (Section of Dermatology), 1927, xx, pp. 61-65; and *Brit. Journ. Derm. and Syph.*, London, 1927, xxxix, pp. 259-262.

different cases, affects the parts of the lower limbs, generally more or less symmetrically,[1] from the upper border of the shoes to a variable distance up the legs; usually the lower third of the limb between the ankle and the knee is specially involved, notably the area above the heel, on both sides of the tendo Achillis, behind the malleoli. (The French speak of it sometimes as being sus-malléolaire). The sufferers are almost exclusively girls and young women, and as one predisposing cause one must assume, I think, a slight endocrine disturbance connected with the female generative organs (sexual hormones).

The condition in question is now being described on the Continent under the heading "erythrocyanosis crurum puellaris (frigida)," but A. V. Karwowski[2] suggests that it should be termed "erythrocyanosis crurum feminarum (frigida)," as it has been known to recur in middle age, and indeed in another female patient (Mrs. F. D.), aged 44, whom I am showing to-day on account of high blood-pressure associated with thrombosis in a retinal arteriole, the same erythrocyanosis of the legs is present in a minor degree. Karwowski holds that cold to the legs and internal generative organs, due to modern clothing, plays an important part in the causation. I would, by the way, here note that I am sure those authors are wrong who assert that the condition never proceeds to definite thickening and hardening of the affected skin.

The condition may (as in the present case) be associated with, but is to be distinguished from, a tendency to ordinary chilblains. It may be associated with, but is also definitely to be distinguished from, "acrocyanosis" or so-called "bad circulation in the extremities" of young persons, *which affects males as well as females.* If a name similar to that of my heading be adopted for the condition, the epithet *frigida* should not be omitted, for it distinguishes the condition from (tuberculous) erythema induratum ("Bazin's disease"), with which severe cases have doubtless often been confused,[3] and from erythromelalgia,[4] which I now regard as a syndrome due to a chronic inflammatory state of the peri-arterial sympathetic nerve-supply to the affected part or parts (one or more toes, a foot, a hand, etc., as the case may be). It is to some extent analogous to chlorosis (which has now become very rare), in so far as it almost only affects youthful females and only those who have an abnormal predisposition to it and in whom the exciting cause (whatever it may be) is at work.[5] The exciting cause in the erythrocyanosis cases is the local action of cold, and the predisposition consists in the *idiosyncrasy* to respond to local exposure to moderate cold in the manner in question and not necessarily or merely by the development of ordinary chilblains.

The present case is that of a young unmarried woman, A. B. L., aged 26, who is "well nourished" and of a "fresh" complexion. The parts chiefly affected are the backs of the ankles, behind the malleoli, above the heels. On these parts are subcutaneous swellings covered by purplish cyanotic skin which are just above the level of the upper border of her shoes. The anterior-internal portion of the lower segment of the left leg, and the anterior-external portion of the lower segment of the right leg are similarly diffusely affected, but to a lesser degree. The somewhat asymmetrical distribution in the present case is unusual, and might possibly be explained on the

1 The term "erythrocyanosis cutis symmetrica" has also been employed.

2 A. V. Karwowski, *Dermat. Wochenschr.*, Leipzig, 1927, lxxxv, p. 1161; see also A. Alexander, "Erythrocyanosis Crurum Puellaris," *ibid.*, 1927, lxxxv, p. 601.

Erythrocyanosis frigida may apparently occasionally be followed by true tuberculous "Bazin's disease."

Cf. F. Parkes Weber, "Erythromelalgia," *Med. Press*, London, 1927, clxxv, p. 266. I have lately had reason to believe that an erythromelalgic condition of a hand, with paroxysms of intense flushing and local heat, may supervene on a previous chronic chilblainy condition, possibly owing to involvement of the peri-arterial sympathetic nerve supply.

5 F. Parkes Weber, "Two Diseases due to Fashion in Clothing," *Brit. Med. Journ.*, 1925 (i), p. 260.

86 Parkes Weber : *Erythrocyanosis Frigida Crurum Puellarum*

supposition that she sits at her work (connected with the making of advertisement cards, etc.) in a slight draught.

The swellings commenced in the autumn of 1926,[1] with itching (as in the early stage of ordinary chilblains) and afterwards pain ; they feel cold to the touch, whereas the swellings in erythema induratum (" Bazin's disease ") feel hot to the touch. She never previously had anything similar, though she had been rather subject to ordinary chilblains of the toes and fingers. Her hands tend to be cold and moist, though she has never had any typical "acrocyanosis," nor Raynaud's symptoms. At present her fingers have been chilblainy since the end of December, 1927. In other respects she has enjoyed good health. Menstruation commenced at 10½ (after an attack of scarlet fever) and has been fairly regular and of moderate amount since then. Her thyroid gland appears to be of normal size. She wears thin woollen stockings, and is careful to avoid traumatism, and never warms her legs at the fire. Wassermann reaction : negative. Pirquet's cuti-reaction for tuberculosis : weakly positive, as it usually is in adults. Nothing abnormal by examination of the thoracic, abdominal and pelvic organs, nor by examination of the eyes, blood and urine. Brachial blood-pressure : systolic, 135 mm. Hg ; diastolic, 85 mm. Hg. Thyroid treatment and intravenous injections of calcium lactate may have temporarily benefited her. The avoidance of sweets, etc., helped her to lose previous excess of fat. Artificial light (ultra-violet) baths make her feel generally better, she thinks. Complete rest in bed has, of course, a very good effect on the local condition of her legs.

The somewhat asymmetrical distribution of the erythrocyanosis in the present case is noteworthy, and, as above stated, I can only suggest that it is possibly due to a slight draught along the floor, from one side, when she is at her work. But I have met with one case in a girl (E. K.), aged 19, in which the condition was unilateral and was limited to the right leg, presumably because the right leg was a *locus minoris resistentiæ*, being smaller than the left leg, probably on account of an attack of infantile paralysis in early life.

Dr. G. M. SLOT said that in this disease there was usually some sexual disorder, or else the blood calcium was low, i.e., 6½, or perhaps 8, instead of the normal 10 or 12. It seemed necessary to activate the calcium, therefore in his cases parathyroid had been given by the mouth in association with calcium intravenously. In two cases, still under treatment, the œdema, which had long been present, had been much reduced.

Tuberculous Pericarditis.

By D. EVAN BEDFORD, M.D.

D. P., FEMALE, aged 18, was admitted to the Middlesex Hospital in August, 1927.

History.—No family history of tuberculosis. Whooping-cough when aged 4 ; tonsils removed when aged 17 ; influenza in October, 1926. Was quite well until three weeks before coming to hospital, when she became feverish and breathless and noticed swelling of the legs. She also complained of pain in the left shoulder.

Examination (August 2, 1927).—She was pale, slightly cyanosed and breathless, and appeared gravely ill. Temperature 101·5° F., respiration 36, pulse 130, regular. Blood-pressure 115/80 mm. Cardiac impulse almost impalpable. Cardiac dullness increased, extending 4½ in. to the left and 2 in. to the right of mid-line. Heart sounds very distant. No pericardial friction audible. Moderate œdema of legs. Lungs : Flattening below right clavicle, and diminished movement of right chest ;

[1] I showed her at the Section of Dermatology on January 20, 1927, *Proc. Roy. Soc. Med.* (Section of Dermatology), 1926-1927, xx, p. 61, case 1.

impairment of percussion note at right apex, and definite dullness at the angle of the right scapula posteriorly, and at the left base. Bronchial breathing at right apex and at right scapular angle. Râles at both apices and all over right lung posteriorly. Pleural friction at left apex. Urine contained albumin.

X-ray examination showed a large pericardial effusion and tuberculous infiltration of both lungs, mainly affecting the right mid-lung field.

Blood-count, September 5, 1928: Red cells 3,420,000, hæmoglobin 68 per cent.; white cells 14,200: polymorphs 70 per cent., lymphocytes 28 per cent., large mononuclears 2 per cent. No sputum was ever obtained.

Pericardial effusion and tuberculous infiltration of the right lung.

Course.—Remittent or intermittent fever continued for twelve weeks, the temperature rising to 103° F. at night and falling several degrees by morning. Tachycardia persisted, rate 115-130. Blood-pressure fell progressively, being 65/40 mm. on August 13, 52/35 mm. on August 17. With improvement a rise of blood-pressure took place, and it was 105/60 mm. in November, 1927. Cardiac dullness increased slightly, especially upwards to the second rib, then gradually diminished. Pericardial friction was heard on several occasions. Signs in the lung diminished, though dullness, bronchial breathing, and râles persisted on the right side. On September 5 and 6, definite rigors occurred and paracentesis of the pericardium was attempted via the fifth right space at the costo-sternal junction. No fluid could be withdrawn through a medium sized needle, and a second attempt was also unsuccessful. No sputum was ever obtained and there was no cough.

88 Evan Bedford : *Tuberculous Pericarditis*

Slow improvement took place and fever subsided after three months except for occasional rises to 100-101°. On October 3, blood-count showed 6,500,000 red cells, hæmoglobin 80 per cent., white cells 15,000.

Skiagrams at intervals showed progressive absorption of pericardial effusion and diminution in size of the heart shadow. *Orthodiagram*, in December, 1927, showed the heart to be almost normal in size, though widening of the heart pedicle persisted. Horizontal diameter, 11·2 cm., mid-line to right border, 4·2 cm.; mid-line to left border, 7 cm. Longitudinal diameter, 14 cm.

The patient left hospital in December, 1927, and now attends as an out-patient. There are signs of infiltration of the right lung, but the heart is practically normal on clinical examination.

Discussion.—Dr. F. PARKES WEBER thought the pericardial fluid was often gradually absorbed as the heart dilated. He thought that as a result of tuberculous pericarditis the pericardium often became adherent, greatly thickened, and sometimes ultimately calcified. In such cases the lower part of both lungs not rarely became adherent from tuberculous "polyserositis," and sometimes great perihepatitis ("Zuckergussleber") and perisplenitis "Zuckergussmilz" developed. The patients might die at about 50 years of age with chronic ascites (which had required frequent paracentesis) and cardiac insufficiency.

Sir HERBERT WATERHOUSE (President) said that he had once seen a case in which he was asked to do paracentesis for tuberculous pericarditis, with marked effusion. Neither a medium-sized needle nor a larger needle produced any fluid; he therefore incised the pericardium, and removed 30 oz. of a glairy, ropy fluid. Its consistency explained its non-appearance when needles were used. He asked why there should be a high temperature with tuberculous peritonitis.

Dr. DOROTHY HARE said she had had one case in which, apparently, the onset of the tuberculous infection had been in the heart. There was a very large pericardial effusion, and on puncturing about 5 c.c. of fluid had come away. It was clear fluid and not thick. The culture grew tubercle bacilli.

Dr. BURRELL said he had seen the case to which Dr. Dorothy Hare referred; there had been fluid in both pleural cavities and in the abdominal cavity, and tubercle bacilli had been found in the fluid from all those sites. Sometimes when one used even a large needle, only a few drops of pus came out. But if one put another needle above, this let in some air, and thereafter the fluid came out quite freely.

A Case of Epithelioma of the Nose and Lip treated by Diathermy Fourteen Years Ago.

By PHILIP TURNER, M.S.

A. M., THEN aged 29, was admitted to Guy's Hospital in March, 1913, for ulceration of the nose and upper lip.

Seven months before admission the inner surface of the left nostril became swollen, ulcerated and painful during the course of a severe cold. The ulcer did not heal, but gradually spread to the cutaneous surface of the left ala nasi and the upper lip. The ulcer was painful and was associated with pain in the forehead.

On admission, there was an extensive ulcer involving the whole of the left ala, the inner surface of the left nostril, and the left half of the upper lip. It was surrounded by an inflamed area, and had a raised hard edge. No enlarged glands could be felt. A diagnosis of epithelioma was made and was confirmed by histological examination.

Operation, March 20, 1913.—The raised, hard edge of the ulcer was excised, and the whole was then treated by diathermy. In order to get a more intensive effect the indifferent electrode was applied to the left side of the neck. This, unfortunately, caused a severe burn which extended down to the deep fascia. A large slough separated, and the granulating wound was skin-grafted three weeks later. The patient was discharged on April 19, and was then using a rubber bougie to prevent contraction of the left nostril.

FIG. 1.—Before treatment.

The patient was readmitted in September, 1913. The wound had then completely healed, but there was, as the result of cicatrization, a severe deformity of the upper lip, resembling a large, wide, incomplete hare-lip. A plastic operation, on the lines of a hare-lip operation, was performed for this, the nostril, which had contracted a good deal, was dilated, and the patient advised to continue with the bougie.

She was lost sight of during the war, and was not seen again until about two weeks ago.

90 Turner: *Epithelioma of Nose and Lip Treated by Diathermy*

There is no sign of any recurrence of the disease, and there is no noticeable deformity of the lip; the left ala nasi is absent, and the left nostril is almost completely stenosed, a small orifice remaining large enough to admit a small probe, through which she can breathe with difficulty. She complains of pain in the forehead when she has a cold, and says that she always has a collection of discharge in the left side of the nose which it is difficult to get rid of on account of the smallness of the nasal orifice.

FIG. 2.—After treatment.

It is proposed next week to perform a plastic operation to increase the size of the left nostril.

Last November the patient was admitted under Mr. Slesinger for pain and swelling of the left foot. She had injured the foot in February, 1927, when it was painful, swollen and bruised, but there was no open wound. The pain and swelling subsided after a fortnight, but recurred a month later and gradually increased until the date of admission. An X-ray examination then showed infective arthritis of the

third metatarso-phalangeal joint. The phalanx was displaced outwards, its base forming a sequestrum. An incision was made over the joint, an abscess opened, and the base of the phalanx and the head of the metatarsal were removed.

Discussion.—Sir HERBERT WATERHOUSE (President) said that with regard to the obstruction in the left nostril, he agreed with the suggestion to make a hole through the septum to facilitate draining through the right nostril.

Dr. E. STOLKIND said that by "surgical diathermy" an almost bloodless operation could be successfully performed in a number of cases.

Addison's Disease.

By ARTHUR G. PHEAR, C.B., M.D.

D. W., AGED 25, female, shorthand typist.

1925.—An attack of jaundice, duration six weeks, in bed two weeks.

November, 1926.—An attack of influenza. Since then, skin has become increasingly dark. No impairment of vigour till summer of 1927, when she noticed that on playing games she became more easily tired than usual.

November, 1927.—Admitted to hospital. Marked pigmentation, generalized, except for scalp and mucous membranes. Most pronounced on face, back of neck, arms, hands and nipples. Blood-pressure: systolic 115, diastolic 65. No symptoms or signs of alimentary tract disorder. No evidence of tuberculosis of lungs or elsewhere.

February, 1928.—Again under observation. Pigmentation now more marked; general colour a deep brown; finger knuckles blackish; lines on palms of hands mapped out by deep pigment. Lining membrane of cheeks now involved. A few small almost black spots on arms and legs.

Has recently suffered from attacks of hiccough, very severe, lasting ten to fifteen minutes; no other gastro-intestinal symptoms. Loss of vigour not marked; has been at work up to date. Blood-pressure 104/50.

Discussion.—Dr. F. PARKES WEBER thought that the case was most likely true Addison's disease, and that pigmentation was often best marked in the less acute cases of Addison's disease. In many genuine cases the suprarenal disease was not tuberculous.

Dr. SLOT said that at Charing Cross Hospital four years ago there had been a case with similar features. In that case the woman had become almost negroid, she also had gastro-intestinal symptoms, and a low blood-pressure. She was in hospital two or three weeks, and then attended the out-patient department for two years, and, as far as he knew now, she was in perfect health. Such a history showed it was not the disease described by Addison.

He thought the prognosis in Dr. Phear's case was good. No treatment tried seemed to influence the pigmentation.

Occlusion of the Left Subclavian Artery.

By C. E. LAKIN, M.D.

E. S., MALE, aged 56. The patient was having dinner on February 22 when he suddenly experienced acute pain in the chest, passing into the left arm. He dropped the fork which he was holding, and his arm became insensitive and almost immovable, and later blue and cold. After a few hours the pain became much less. His doctor,

92 Lakin: *Occlusion of Left Subclavian Artery;* Cassidy: *Neurosis*

who was called in, could find no pulsation in the left radial, brachial, axillary or subclavian arteries. The patient went to bed, but feeling better next day, got up, but the arm again became blue and painful, and he was recommended to remain in bed. There was a slight rise in temperature for the first three or four days, since which it has remained normal. A collateral circulation has become established, and there has been no gangrene. Fullness can be detected in the left supraclavicular region. The Wassermann reaction is negative.

Discussion.—Dr. E. STOLKIND said that he thought this was a case of syphilitic aortitis with subsequent aortic aneurysm. The negative Wassermann did not exclude syphilitic aortitis; which in adults over forty was frequently accompanied by arteriosclerosis of the aorta.[1] The occlusion of the left subclavian artery, so far as it was not caused by thrombus or embolus, might be due to syphilitic cicatrized and pitted patches in the mouth of the artery; he (Dr. Stolkind) had observed this in some of his own cases.

Dr. EVAN BEDFORD said he disagreed with Dr. Stolkind that there was syphilitic aortitis, because the skiagram of the aorta was typical of atheroma. Cases of occlusion of the subclavian have been described; occasionally it was due to syphilitic arteritis, but in the majority of cases it was caused by atheroma, with or without thrombosis. Dr. Lakin's case seemed to be an example of atheromatous occlusion of the subclavian with a terminal thrombosis. If embolism had occurred, there would almost certainly have been serious circulatory disturbances in the arm and probably gangrene. When occlusion was gradual, compensatory collateral circulation developed slowly so that final complete occlusion became less serious. In thrombotic occlusion the onset of symptoms was often sudden, as, for example, in coronary thrombosis, in which the onset of pain might be sudden and dramatic.

Vasomotor Neurosis.

By MAURICE A. CASSIDY, M.D.[2]

THIS patient is a man, aged 35, a farmer. English parentage, no Jewish relatives. Always a light smoker (five cigarettes a day); never had venereal disease or other serious illness. He complains that when his legs hang down, or if he walks far, his feet become discoloured and painful and discharge. Symptoms began when he was aged 20, as a pricking sensation in the feet, across the instep in front of the heel, brought on by pendency or by exertion. This sensation increased to the point of pain so that he was obliged to stop walking, and then the pain would cease. During the attack the feet became mottled and swollen. These symptoms were promptly relieved by recumbency. At first the symptoms were slight and did not trouble him much. He joined the Army in 1914, but during training he was constantly in and out of hospital, and was finally invalided out in 1916. Subsequently he joined the Air Force, where his feet did not trouble him so much, nor was he seriously incapacitated during the years immediately after the War. During the last three years or so his symptoms have been increasingly severe; some days he may be able to walk three or four miles, but at other times he can with difficulty get about at all owing to the severity of the pain induced by walking. His symptoms are always worse during wet weather; with a high barometer and dry weather he is at his best. His hands are always cold and blue, but apart from this, give him no trouble; his fingers never "go dead" nor have there been any sore spots on the hand. Small ulcerated areas frequently appear on the feet, chiefly in the neighbourhood of the ankle, never on the toes. The skin of the feet "discharges" during the attacks, crusts being formed as the discharge dries.

1 "The Diagnosis of Syphilitic Aortitis," *Med. Press and Circ.*, 1921, clxiii, 9.
2 Report postponed from the Meeting held on December 9, 1927.

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On examination: he is a healthy-looking man with no evidence of any psychoneurotic taint. When the feet are pendent they become at first pink, then more and more deeply cyanosed, ending up in a mottled state of pink and purple hue. The blanching produced by pressure reappears rapidly when pressure is removed. At the same time there is some swelling of the veins, and the dorsalis pedis and posterior tibial pulses disappear, though these pulses are easily felt when the legs are horizontal. There is some pigmentation of the skin of the feet and well-marked trophic changes of the skin and nails. There is no evidence of any loss of tissue at the tips of the toes. On elevation of the limb the colour disappears, but there is no ischæmic blanching. The brachial arteries are normal for his age. Blood-pressure 140/80. Wassermann reaction negative. Blood-count: 6,325,000 red cells; hæmoglobin, 88 per cent.; white cells 2,920: polymorphs, 54 per cent.; C.G.O. 3 per cent.; small lymphocytes, 25 per cent.; large lymphocytes, 10 per cent.; large hyalines, 7 per cent.; basophils, 1 per cent.

Blood-sugar Curve.—Normal.

Electro-cardiogram.—Normal.

Family History.—His father and one brother have suffered from precisely similar symptoms; the father is living, aged 85, and has had very little trouble with his feet during the last ten years. One brother died of pneumonia at the age of 39, having been similarly affected. Two sisters have cold hands and feet but are otherwise normal, and two other brothers and one sister have normal circulation.

The diagnostic possibilities seem to be: (1) erythromelalgia; (2) Raynaud's disease; (3) thrombo-angiitis obliterans.

Cases of thrombo-angiitis obliterans have been recorded in which pain, redness, and trophic changes persisted for years before the onset of gangrene, but in this case the family history and the variation in the radial and posterior tibial pulses seems to point strongly towards a vasomotor neurosis. The variation in the pulse is suggestive of Raynaud's disease, but there is no history of paroxysmal local syncope, nor, during a period of fifteen years, has there been any definite loss of tissue. Hyperidrosis, redness, swelling and pain associated with pendency, point towards erythromelalgia, and in this condition trophic changes in the skin may occur; but exaggeration rather than obliteration of the pulses would be expected during the attacks. In spite of the normal blood-sugar curve it is proposed to try the effect of insulin treatment in this case.

Discussion.—Dr. F. PARKES WEBER thought that the case was one, not of erythromelalgia, but of a particular type of constitutional circulatory defect in the lower extremities, characterized especially by livid patches below the malleoli, with a tendency to the formation of chronic ulcers in those situations. The especially interesting point in Dr. Cassidy's case was the familial history. There was in the patient doubtless a constitutional bilateral defect in the development of the arteries of the lower limbs, with some degree of actual stenosis (insufficient diameter of the arteries). A similar condition of one foot was present in a young woman (A. W.), aged 30, whom he (Dr. Weber) had shown at the meeting on February 11, 1927 (*Proceedings*, 1927, xx, p. 46), but it was in the left foot only, and connected with an apparently acquired condition of chronic arterial ischæmia in the left lower limb.

In the present case, the manifestation of the constitutional defective circulation was very striking, but the condition might be compared with the much commoner conditions which were classified under such headings as "acrocyanosis."

Dr. J. POYNTON asked whether Dr. Cassidy and Dr. Parkes Weber thought these cases were allied to those which occurred in some young women who during cold weather wore unduly thin clothes and developed blueness, ulcerations and pain of great obstinacy in the lower limbs.

Dr. A. P. CAWADIAS said this case could not be a real erythromelalgia or Weir Mitchell's syndrome, because in real erythromelalgia there was no obstruction in arterial circulation of

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the extremities in the intervals of the attacks, and because during the attacks there was throbbing—with intense vasodilatation—of large and small arteries and of capillaries. This patient had obliterating arteritis with secondary erythromelia due to reactionary vasodilatation of the capillaries. Obliterating arteritis belonged to the class of thrombo angiitis obliterans, and this case showed that such thrombo angiitis obliterans was not, as was generally believed, found only in Polish Jews, but also among Englishmen, and in French, Greeks and Egyptians.

Insulin treatment as advised by Vaquez and Yacoel should be tried. Insulin acted principally on the vasomotor element which was of endocrine origin.

He (Dr. Cawadias) had obtained good results by the use of diathermy and intramuscular lipiodol injections, and, principally, by sending his patients to Royat. The Royat waters, rich in carbon dioxide, acted also on the vasomotor element in the disease.

Section of Dermatology.

President—Dr. J. M. H. MacLeod.

[*February* 16, 1928.]

CASES.

Leukæmic Erythrodermia.

By H. MacCormac, C.B.E., M.D.

The patient exhibited, a man, aged 27, presents the uncommon variety of leukæmia accompanied by erythrodermia. His general health previous to the onset of the present condition six years ago had been good. In 1921 he had a general itching of the skin, accompanied by excessive sweating. This condition continued for about three months, and then a weeping eruption developed in the axillæ. Following this, the skin became universally red and moist, boils appeared on the head and back, the hair fell out freely, and there was a purulent discharge from the ears and eyes. Early in 1922 he was admitted to a local hospital, where he so far recovered that he was able to leave after twelve weeks, with only a slight "weeping" condition on the chin. Later there was a gradual reappearance of the eczematous eruption on the head and in the groins; this improved with X-ray treatment. In 1926 he was treated with ultra-violet light applied to the whole body, and following this his skin peeled freely, and a general erythema developed, which has persisted to the present time. The patient was seen in December, 1926, by Dr. Mannix, who recognized the nature of the condition, and confirmed the diagnosis of leukæmia by a blood-count. I am much indebted to Dr. Mannix for allowing me to quote the report then made to him by Dr. Knyvett Gordon:—

Red Cells.

Total count	6,320,000 per c.mm.
Hæmoglobin	100 per cent.
Colour index	0·8.

White Cells.

Total count	88,400 per c.mm.
Polymorphs	10 per cent.
Large lymphocytes	20 per cent.
Small lymphocytes	50 per cent.
Hyalines	3 per cent.
Eosinophiles	2 per cent.
Myelocytes	15 per cent.

Histological examination of a piece of skin showed dense infiltration of the corium with large and small lymphocytes and lymphoblastic plasma cells. The patient was then sent to the Radium Institute, where the enlarged glands in the groins were treated. As a result these glands diminished in size, but the erythrodermia remained unaltered.

I first saw the patient in December, 1927; the skin of the entire body and face was of a light red colour, with a fine desquamation. Over the trunk, upper arms, buttocks and thighs, there were well defined areas of patchy and reticular staining of deep red, from which the colour could only be partially removed by pressure. These darker red areas have increased since the patient first came under observation. The legs were swollen and their surfaces moist. The palms and soles were hyperkeratotic, and the nails raised at their free margins by a heaping up of scaly material. No enlargement of the spleen could be detected, but the glands in the

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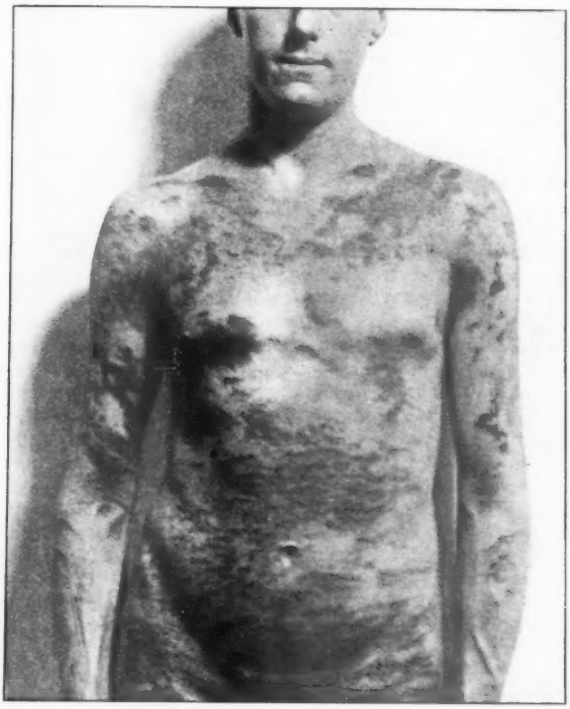


groins, axillæ and left pectoral region were distinctly palpable. In the groins this enlargement has recently become much more pronounced. The patient complained of general itching. A radioscopic examination revealed no glandular enlargement in the thorax. The blood, examined in December, 1927, showed the following changes:—

Red Cells.

Total count	4,930,000
Hæmoglobin	84 per cent.
Colour index	0·86

White Cells.

Total count	19,200 per c.mm.
Polymorphs	20 per cent.
Lymphocytes	77 per cent.
Mononuclears	0·5 per cent.
Eosinophiles	2·5 per cent.

A section of skin showed an infiltration of the dermis with masses of cells with a deeply staining nucleus, closely resembling large lymphocytes.

This case corresponds in its clinical and histological features to those recorded by Sequeira and Panton, in the *Quarterly Journal of Medicine* for 1924-5, under

the name of lymphoblastic erythrodermia. They describe in their paper five examples of this rare form of leukæmia. In only one of their cases—that communicated to them by Dr. W. H. Brown—was any eczematous condition of the skin noted, the ears being affected. Sequeira and Panton consider the clinical features and the blood-picture to be characteristic of the group as a whole, and they lay emphasis on generalized redness of the skin as the most pronounced physical sign, with which there are associated characteristic changes in the white cells consisting of a relative and absolute increase in the lymphocytes. Although these features may constitute strong presumptive evidence of the nature of the disease, they do not appear to be conclusive, for Dr. MacLeod and Dr. Wigley showed at the meeting of this Section in October, 1924, a similar case which, nevertheless, eventually came to be regarded as an example of lymphadenoma with erythrodermia, as reported by Dr. MacLeod in the *British Journal of Dermatology* in the following year.

The expectation of life in this condition appears to correspond with that of leukæmia in general. In Sequeira and Panton's series, three of the patients are stated to have survived three or four years. In my case the duration of life has been unusually long, as the first symptoms of the disease developed in 1921, and the patient is at the present time in surprisingly good health.

Discussion.—Dr. F. PARKES WEBER agreed that the case was of the same class as those published by Sequeira and Panton under the heading "Lymphoblastic Erythrodermia." But on three or four ocasions when cases under this heading were shown to the Section he had maintained that they were forms of chronic lymphatic leukæmia and that they were nothing more nor less than examples of the condition which was first described by Kaposi under the name "Lymphodermia perniciosa."[1] The first describer of a disease nearly always included some other conditions under his original heading; but that was no reason why he should not be given the credit of the discovery of a new class of cases. Kaposi said that before death these patients developed a blood-picture of lymphatic leukæmia. The patients who had been shown at the Royal Society of Medicine seemed to have lived a considerable time after the changes first appeared in the blood.

It was acknowledged that occasionally (though very rarely) one might get general erythrodermia in Hodgkin's disease (lymphogranulomatosis maligna). Dr. Colcott Fox had told him (the speaker) that similar erythrodermia had been noted in tuberculous disease of lymphatic glands. He (Dr. Weber) suggested that the case or cases referred to by Dr. Colcott Fox might have been examples of tuberculosis associated with Hodgkin's disease.

Dr. J. M. H. MACLEOD (President) said that the case to which Dr. MacCormac had referred, shown by himself and Dr. Wigley, had been eventually considered to be one of Hodgkin's disease. It seemed that similar clinical appearances could occur in connexion with a number of blood conditions. He had seen scaly erythrodermia in Dr. Sequeira's lymphoblastic cases, in aleukæmic leukæmia, in lymphatic leukæmia, and in Hodgkin's disease. The only type of case in which he had never seen erythrodermia of this type was spleno-myelogenous leukæmia. He had also seen plum-coloured nodules, similar to those of spleno-myelogenous leukæmia, in connexion with Hodgkin's disease. It was difficult in these cases to surmise the underlying blood conditions from the appearance of the skin.

Dr. S. E. DORE said it was difficult to distinguish some of these cases from cases of parapsoriasis. For some time a man was under his care who had previously been under that of Dr. Cranston Low, with a diagnosis of parapsoriasis. When he (the speaker) saw the case the blood-count was normal, and there was an eruption not unlike that in Dr. MacCormac's case. Eventually the eruption changed its character and was followed by much thickening of the skin. The patient subsequently went to the London Hospital, where he was under Dr. Sequeira as a case of acute lymphoblastic erythrodermia. It seemed possible that some of the cases diagnosed as parapsoriasis might be examples of this condition before the blood changes took place.

[1] F. Parkes Weber, in discussion, *Proc. Roy. Soc. Med.*, Section of Dermatology, 1923, xvi, p. 105; 1919, xii, p. 55 and p. 56.

Dr. PARKES WEBER said he understood that in the case (a man, G. S., aged 60) referred to by Dr. Dore the post-mortem examination had revealed definite inter-acinous infiltration with leukæmic cells in the liver.

Dr. MACCORMAC (in reply) said he thought a distinction should be drawn between the erythrodermia associated with Hodgkin's disease and that accompanying leukæmia, and on this ground the terminology employed by Sequeira and Panton (lymphoblastic erythrodermia) was open to criticism. Although leukæmia and lymphadenoma might give rise to similar manifestations on the skin, since most authorities now regarded these diseases as separate entities, it was more important to emphasize the underlying causal disease in the nomenclature.

Acanthosis Nigricans.

By M. SYDNEY THOMSON, M.D.

THIS boy, aged 13, first attended hospital on January 20, 1928. His father, who is an intelligent man, insists that the eruption had made its appearance for the first time on January 14. It was then noticed that there was a "chapping" of the back of the neck and of the hands. Afterwards gradual extension occurred over the various areas of the body until a few days ago, when there began to be apparent a slight but definite improvement in the condition of the hands.

In view of his present condition I admit that it seems incredible that the history can be so short. Nevertheless, I believe his father's statement to be true, for although the whole of the legs now shows extensive scaling, this picture has appeared during his stay in hospital; at his first attendance there was only a little hyperkeratosis over the knees and insteps. Also, it must be pointed out that a considerable loss of pigment has taken place during the last week, whilst erythema has appeared for the first time. The following is a description of the case, with stress laid on the points which led to the diagnosis of acanthosis nigricans a month ago.

There is a dusky brown discoloration of the nape of the neck, together with a certain amount of superficial fissuring and scaling. Similar changes have occurred over the knees, upper parts of the thighs, in the axillæ and over the hands and feet. In the region of the arm-pits it can be seen that the light brown pigmentation extends for at least half an inch beyond the scaling in the centre. In the same place, too, an accentuation of the natural lines of cleavage can be clearly seen. It might be said that this exaggeration almost suggests very tiny papillomata. There is definite thickening of the skin of the palms, together with a roughened and hard surface. Finally, reference must be made to the dark brown discoloration which involved the umbilicus and surrounded it for a quarter of an inch. This was accompanied only by slight scaling. The mucous membranes are nowhere affected.

The family history contains no suggestive points, neither does the boy's own history help. Indeed, he has apparently never suffered from any gastro-intestinal disturbance whatever, nor was the onset of this eruption preceded by any prodromal symptoms. There is no story of exposure to either extreme heat or cold. The fact that he has always been liable to suffer from chapped hands and knees may possibly lend some support to the suggestion made by Dr. Parkes Weber in 1920 that patients who show thickening of the skin round the ankles in cold weather may, in reality, be mild examples of a similar condition.

The following results of various pathological investigations were obtained. The Wassermann reaction is negative. Chemical and bacteriological examinations of the urine and fæces revealed nothing abnormal. There is no melanogenuria. The blood-count, the basal metabolic rate and the hepatic function tests are all normal, as also is Loewi's test. The blood-sugar curve shows a normal, but low outline. As

this type has been observed in some hypothyroid and pituitary cases, the test was repeated after the administration of atropine, when exactly the same curve was obtained. It seems probable that there is a real increase in carbohydrate tolerance, for an opaque meal proved that the stomach empties normally. At the same time it was observed that the sigmoid colon is long and redundant, thus forming an excellent focus from which toxæmia could arise. Radiographs were also taken of the abdomen and of the skull, but these showed no calcareous glands, changes in the pituitary fossa, etc. Dr. East kindly examined the boy, but could find no abnormal signs in the chest, abdomen, or central nervous system. The systolic and diastolic blood-pressures are 120 and 80 mm. Hg respectively.

Histological examination of a piece of skin removed from the anterior fold of the left axilla, seems to support the diagnosis of acanthosis nigricans. There is marked hyperkeratosis and the granular layer is thickened in some places to the depth of four cells. Acanthosis is present in some areas, but is not marked. The amount of melanin present in the basal layer and adjacent epithelial cells is small, but it can be detected in unstained sections. The interpapillary processes show slight proliferation, whilst the surface of the tissue occasionally demonstrates the exaggeration of the normal ridging of the skin. These protuberances have not, however, progressed so far as to be definite papillomata, for they are only very small and contain no core of true skin. There is a slight perivascular infiltration, but no mast cells are present.

Discussion.—Dr. F. PARKES WEBER said that in a discussion some years ago[1] he alluded to a warty, nutmeg-graterlike, dark thickening of parts of the skin, notably about the ankles, during cold seasons, which disappeared with the onset of warmer weather. He regarded such cases as very *minor varieties of acanthosis nigricans*, and suspected that they were connected with some form of chronic intestinal toxæmia.

Dr. Weber thought there was an acanthotic process, associated with darkening of the affected skin, which must be distinguished from typical cases of "acanthosis nigricans."[2]

Dr. H. W. BARBER said that Dr. Thomson would agree that the diagnosis of acanthosis nigricans could not hold good. His (the speaker's) view was that the diagnosis lay between pityriasis rubra pilaris and, possibly, arsenical poisoning.

Dr. GRAHAM LITTLE said he had seen three cases of pityriasis rubra pilaris which followed upon the use of arsenic. He agreed with Dr. Barber's suggestion.

Dr. J. M. H. MACLEOD (President) said he did not think Dr. Thomson now adhered to the diagnosis of acanthosis nigricans in his case. In the first place, the patient was a male, and that condition was twice as frequent in women as in men; moreover, it was very rare in young people. Some years ago he had analysed the records of a large series of cases of acanthosis nigricans, and in only 30 per cent. of them was there any associated malignant disease, though admittedly, in a certain number of them, there was no opportunity of proving or disproving this association.

Postscript (February 24, 1928).—No trace of arsenic has been detected in specimens of urine, hair and scales taken from various parts of the body. As no follicular papules have been found clinically, very careful examination has been made of numerous sections of the skin. The hyperkeratosis, etc., is general and nowhere has there been seen any disproportionate or particular involvement of the follicles. Taking all the facts into consideration I regard the case as one of the minor varieties of acanthosis nigricans mentioned by Dr. Parkes Weber.—M. S. T.

[1] Discussion on a case of Dr. H. MacCormac, *Proc. Roy. Soc. Med.*, 1920, xiii (Sect. Derm.), 115-117.

[2] Cf. abstract of Guérin's monograph on what he calls "infectious acanthomata," *Presse médicale*, Paris, 1926, xxxiv, p. 1322.

Two Cases of Sarcoid.

By H. W. Barber, M.B.

(I) N. F., aged 34, a married woman, gives no history of any important illness, and no definite evidence of tubercular infection is forthcoming.

The cutaneous lesions appeared first about a year ago (February, 1927) on the left leg, and on the face in June, 1927.

This form of sarcoid is, I think, uncommon. It occurs as flat, slightly raised plaques of varying size, yellowish in colour and lupoid in appearance; the tint is of a lighter, more vivid yellow than that of lupus vulgaris or of the commoner variety of sarcoid. The plaques undergo spontaneous involution in the central portion, with definite scarring, so that they may present a central depressed area of atrophy with a raised ring of yellowish sarcoid tissue at the periphery, at first sight not unlike the appearance of a superficial basal-celled carcinoma.

I remember several years ago seeing a patient—a South African woman—who had already consulted and puzzled two other London dermatologists, one of whom I believe (before he saw a microscopical section) diagnosed multiple basal-celled carcinoma. There were a large number of these flat yellow plaques, some of them very large, situated chiefly on the forehead, scalp, neck and back. There was central atrophy with quite deep scarring in the larger plaques. Microscopical sections confirmed the diagnosis of sarcoid. Another very similar case, a patient who also had iritis, was under my care two years ago.

The present patient has several plaques of varying size on her cheeks, forehead, and behind the left ear; one is also present on the right elbow, two on the left knee, and one on the back from which the biopsy was made.

The von Pirquet reaction is completely negative to both human and bovine tuberculin. The Wassermann reaction is also negative. A radiogram of the chest, taken by Dr. Lindsay Locke, shows rather dense hilus shadows, with fine striation spreading out in the lungs. The microscopical sections exhibited are typical of sarcoid.

(II) A. H., female, aged 52.—No history indicative of tubercular infection. The eruption began in May, 1927, on the forehead, and shortly afterwards a flat lesion appeared on the right side of the nose.

There are present on the forehead, discrete, raised reddish-brown nodules, but, since the patient has been under treatment with krysolgan, several have disappeared. She has had eight injections up to date. On the right side of the nose is a flat plaque, made up of several confluent nodules.

The von Pirquet reaction, as in the first case, is completely negative, as is also the Wassermann reaction. A radiogram of the chest shows little that can be considered abnormal.

Discussion.—Dr. H. C. Semon said that he had had under his care a very similar example of a nodular eruption on the face and forehead of a woman aged about 40. The tests described by Dr. Barber were done in this case, and the von Pirquet reaction was one of the most violent he had seen. He knew Schaumann laid great stress on the absence of the von Pirquet in true sarcoid, he would therefore be glad to know if the diagnosis could be upheld. The effect of two small injections of krysolgan was dramatic, for the eruption had almost completely cleared up. A swelling of the pre-auricular glands on both sides which followed suggested a direct local action on the nodules, and subsequent glandular absorption.

Dr. F. Parkes Weber asked whether Dr. Barber meant that the presence of sarcoid inhibited the appearance of a positive von Pirquet reaction, such as almost normally appeared in apparently healthy adults. He thought that in adults supposed to have Hodgkin's disease it was nevertheless worth while taking the von Pirquet reaction, as occasionally it was negative, and that strengthened the diagnosis of Hodgkin's disease.

Dr. BARBER (in reply to Dr. Semon) said that if in Dr. Semon's case the von Pirquet reaction was positive, Schaumann would at once say that sarcoid was excluded.

In reply to Dr. Parkes Weber: Schaumann's idea was that in true cutaneous sarcoid the von Pirquet reaction was more negative than in the apparently healthy, normal adult, but that, if pulmonary tuberculosis developed in cases of sarcoid, the cutaneous lesions automatically disappeared and the von Pirquet reaction became strongly positive.

Adenoma Sebaceum.

Shown by R. T. BRAIN, M.D. (for Dr. W. J. O'DONOVAN).

C. J., MALE, aged 23, has the typical eruption of adenoma sebaceum. The case is of special interest because the patient's mother and sister have a similar condition.

The sister was admitted to a mental hospital in infancy. There do not appear to be any signs of mental defect in C. J. or his mother.

Ulceration of Nipple: ? Paget's Disease.

By E. G. GRAHAM LITTLE, M.D.

THE patient, a married woman, aged 56, was sent to me by my colleague Dr. Leonard Phillips, from the Soho Hospital, with the diagnosis here suggested. She shows chronic ulceration of the right nipple of some twelve months' duration. The skin surrounding the nipple is intact. There is some thickening at the base of the nipple as compared with the other side, but no involvement of mammary tissue can be detected by palpation, and there are no enlarged glands in the axilla.

Nodular Infiltrations in Subcutaneous Tissue: Case for Diagnosis.

By E. G. GRAHAM LITTLE, M.D.

THE patient, a married woman, aged 40, shows deep, nodular, circumscribed infiltrations in the subcutaneous tissue, dating from twelve months ago, and found chiefly upon the trunk, the buttocks and the arms. They are distinctly tender upon pressure, but are also painful without pressure. There is a patch of dark brown pigmentation, measuring about 2 in. by 1 in., upon the left upper arm. There are no molluscum fibrosum tumours. The case is probably an early example of adiposis dolorosa.

Lichen Planus of Rare Type.

By E. G. GRAHAM LITTLE, M.D.

PATIENT, male, aged 55.

Both forearms from just above the elbow to the wrist are occupied by an eruption consisting of small papules, purplish-blue in colour, arranged in a fenestrated way, enclosing meshes of about a quarter of an inch in diameter, and in some cases in lines, not in meshes. The redness blanches almost, but not quite completely, on glass-pressure. On the dorsum of the hands a more follicular arrangement is present (not meshes), and upon the upper and inner surfaces of the thighs there are discrete, small, follicular papules. Upon the glans penis there are a number of dark brown pigmented areas, from one-sixteenth to one-eighth of an inch in diameter. Upon the left side of the scrotum there is a broad band of lichenification extending from the base of the penis to the lower surface of the scrotum. There are small patches of scaliness upon the right side of the scrotum. On the mucous membrane of both sides of the mouth, on the lower lip, and at the base of the tongue there are white patches like lichen planus. Completely edentulous. Never had X-ray treatment.

Dr. J. M. H. MACLEOD (President) said he agreed that the condition was lichen planus, but this was a very unusual case. He had a picture of a leg showing the same kind of lesions, and in that case, as in the present one, the lichen planus of the mouth was characteristic.

Chronic Stagnatory Erythema of the Legs Treated by Arterial Sympathectomy.

By S. E. DORE, M.D.

THIS patient, a woman aged 61, has suffered for fifteen years from cyanotic erythema of the lower third of the legs. During the winter months the condition is greatly accentuated, causing severe pain, and accompanied by the eruption of bullæ. Numerous methods of treatment having been tried, she was referred to Mr. Rock Carling, who performed arterial sympathectomy in the summer of 1927. The condition has not improved since the operation, and during the cold weather was as severe as it had been previously.

The case is exhibited to show that peri-arterial sympathectomy is apparently valueless in these cases. This is the view of Mr. Rock Carling, who has operated on two other cases of a similar character without any permanently beneficial results.

Dr. F. PARKES WEBER said that from recently published accounts of cases it seemed probable that the prevalent name for the condition would be something like "erythrocyanosis frigida crurum puellarum," but Karwowski suggested that "puellarum" should be replaced by "feminarum," as the condition had been known to recur in middle age. Karwowski[1] holds that cold to the legs and internal generative organs, due to modern clothing, plays an important part in the causation. Dr. Weber believed that cold to the legs was the exciting factor (from which sufferers should be protected), but that only those girls and women suffered who had the special idiosyncrasy of reacting towards cold in that way.[2] Not all suffered who wore thin stockings and short dresses.

Specimen: Microscopical Section showing Adeno-carcinoma of Sweat-gland.

By J. H. T. DAVIES, M.B.

THIS specimen came from the anterior part of the scalp of a girl aged 18. She had noticed a sore place for six months.

On examination, there was a pedunculated swelling the size of a pea, bathed in pus. It was soft, red, slightly lobulated and it bled readily. Separated from the base of its stalk by about a quarter of an inch of scar, were two or three granulations, in all three-eighths of an inch in diameter. The tumour, which appeared to be a granuloma pyogenicum, was snipped off, and the base, which seemed to have the typical pale indurated edge, was cauterized with a drop of phenol.

The histology at the time was not obvious.

Failure to heal on the part of the ulcer after some eight weeks' treatment, including a pastille dose of X-rays, which had no effect at all, led to its excision. In both sections there is definite formation of acini by the cells of the growth, and in the second section, reasonable evidence of malignancy.

Dr. H. MACCORMAC said that some parts of the section resembled basal-celled carcinoma.

Dr. J. M. H. MACLEOD (President) said that he agreed with Dr. MacCormac, but a diagnosis could not be made from this specimen.

[1] Karwowski, *Dermat. Wochenschr.*, Leipzig, 1927, lxxxv, p. 1161.
[2] F. Parkes Weber, *Brit. Journ. Derm. and Syph.*, London, 1925, xxxvii, p. 259, and 1927, xxxix, p. 259.

Section of Pathology.

President—Dr. J. McINTOSH.

[*March* 20, 1928.]

Some Observations on Pyuria.

By CUTHBERT DUKES.

THE discursive title "Some Observations on Pyuria," allows me to include in my remarks three different aspects of pyuria: (1) A numerical definition of pyuria, (2) a suggested classification of pyuria, and (3) the course of urinary infection following catheterization.

(1) *Numerical Definition of Pyuria.*

The presence or absence of pus is one of the most important of the urine tests, yet hitherto there has not been any accepted standard for defining the limits of normal and pathological excretion of leucocytes in the urine.

These limits cannot easily be determined accurately if the centrifuge or sedimentation methods are relied on for preparing urine for microscopic examination. Being dissatisfied with these, I have recently described a new method for the examination of urine for pus which consists in counting the urinary leucocytes of a fresh, uncentrifuged specimen in a cell-chamber similar to the Fuchs-Rosenthal cell-chamber devised for cell-counts in the cerebro-spinal fluid. The results are expressed as the number of leucocytes per cubic millimetre of urine. This simple test can be carried out in two or three minutes, and the only apparatus needed is a ruled slide and cover slip, a microscope with a movable stage and a $\frac{1}{3}$-in. objective.

I have been using this method for nearly two years, and applied it to more than 1,000 specimens of urine.

Having before us the figures expressing the number of leucocytes per c.mm. of urine of several hundred healthy men and women, and of a corresponding number of cases of urinary sepsis, I suggest the following limits for what shall be considered normal and pathological excretion of leucocytes: (1) Less than ten leucocytes per c.mm. may be regarded as normal. (2) Between ten and 100 leucocytes per c.mm. should be spoken of as "excess of leucocytes" (see fig. 1). (3) Above 100 leucocytes per c.mm. is definitely pathological and should be labelled pyuria.

These definitions are expressed diagrammatically in fig. 1. By "normal" is meant consistent with a healthy genito-urinary tract. The significance of counts within the zone of "excess of leucocytes" becomes less or greater according as the lower or upper limit is approached. Little attention need be paid to counts of ten to fifty in women, though in men such a record should be treated more seriously. The diagnosis "excess of leucocytes" is rather unsatisfactory, and my plan for these cases is to repeat the test after giving strict instruction to avoid external contamination during collection of the specimen, since experience has shown that the commonest cause of excess of leucocytes in women is a septic condition of the external genitals.

These generalizations are as far as I propose to go at present, more detailed analysis being postponed until the numbers in each group are large enough to make statistical analysis of value. In the meantime, the following evidence is offered in support of these generalizations. In the first 700 cases[1] the average for normal

[1] Cuthbert Dukes, "The Examination of the Urine for Pus," *Brit. Med. Journ.*, 1928 (i), 391-393.

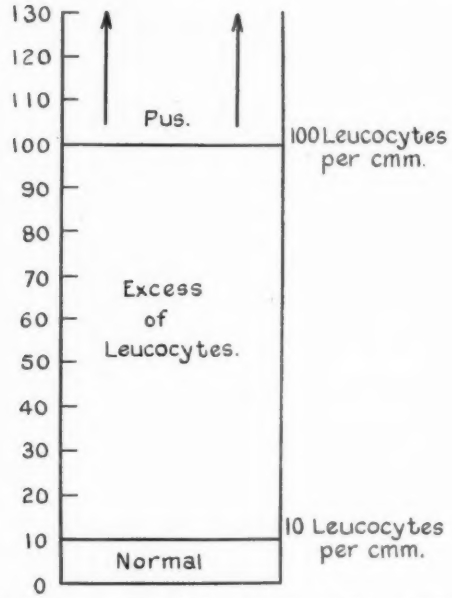


FIG. 1.—Diagram to illustrate definition of "excess of leucocytes" and "pus."

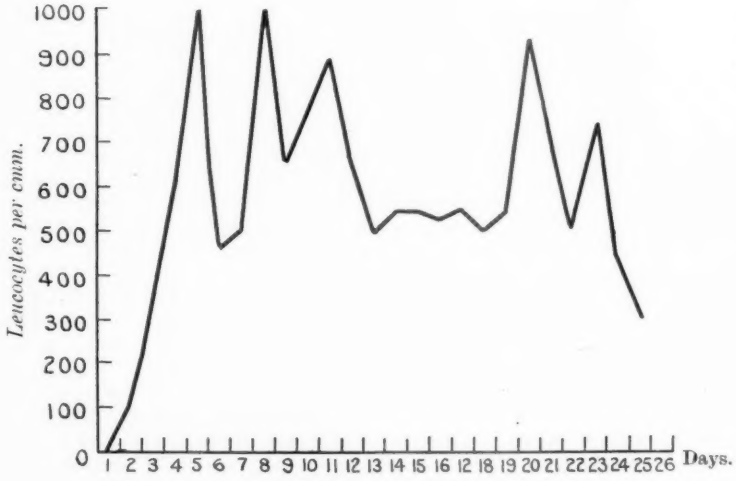


FIG. 2.—Continued pyuria due to *B. coli* infection of the bladder.

women was 7·1 leucocytes per c.mm., and for normal men 2·1 leucocytes per c.mm. The last 300 cases gave an average for healthy women of 5·4 leucocytes per c.mm., and for men of 1·6 leucocytes per c.mm. In support of my definition of pyuria as urine containing more than 100 leucocytes per c.mm., I can say that I have never known a case of persistent urinary sepsis in which the leucocyte count was less than 100 cells per c.mm. More than 500 cells per c.mm. are usually present in acute urinary infections, and in most chronic infections between 100 and 500 cells per c.mm. The intermediate class between 10 and 100, which I have called "excess of leucocytes," is a useful neutral zone between the normal and pathological. Such a generous margin is necessary because the limits of excretion of leucocytes by normal people cover a fairly wide range, and because of variation in degree of dilution depending on the amount of fluid secreted by the kidney per hour.

Variations in the degree of dilution, however, may be reduced to a minimum if the cell-count be made on the first specimen passed in the morning as I have already suggested.

(2) *Classification of Pyuria.*

One great advantage of the cell-count method of estimating pyuria is that it provides a daily record of the excretion of leucocytes, the increase or decrease of output

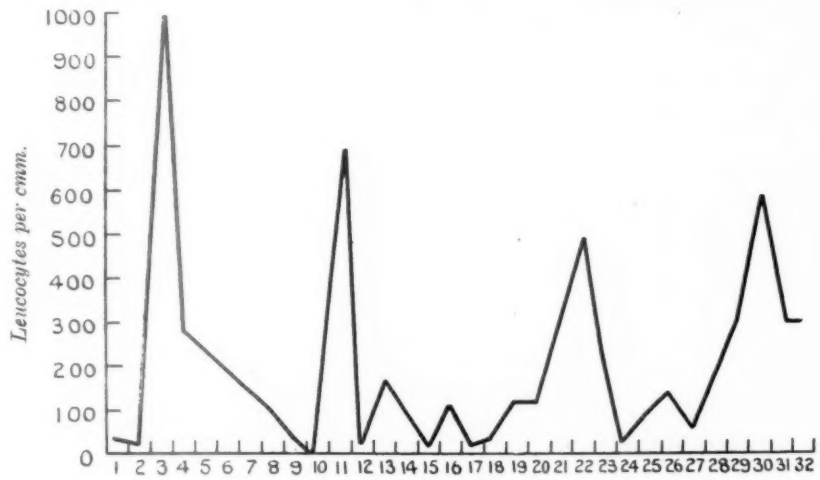


FIG. 3.—Intermittent pyuria.

being registered as easily as the temperature or pulse. Using the method in this way I have learnt to distinguish two types of pyuria, the continued and the intermittent, which may be defined as follows. In continued pyuria the pus is present every day; although there may be daily variations, there are always more than 100 leucocytes per c.mm. in the first morning specimen of urine. In intermittent pyuria the pus is present on one day and absent on another: it reappears at fairly regular intervals, alternating with periods when the urine is free from pus. These two types of pyuria are illustrated in figs. 2 and 3. Continuous and intermittent pyuria are probably characteristic of different clinical conditions, and this classification may be

of some use in diagnosing the cause of pyuria in a patient. I have some evidence in favour of this but it is not yet sufficient to be of practical value. In the meantime I draw your attention to intermittent pyuria because it shows that when disease of the genito-urinary tract is suspected one examination is not sufficient to disprove the existence of pyuria.

(3) *Urinary Sepsis following Catheterization.*

In studying the origin and course of urinary infections I have been fortunate in discovering close at hand a form of urinary sepsis developing at a predictable date in men free from previous disease of the genito-urinary tract. As part of the routine operation for excision of cancer of the rectum, it is the custom of the surgeon to tie-in a catheter and leave it in position after the operation for three to five days, the urine being released at regular intervals. I found that pyuria invariably developed in these patients. I have made daily leucocyte counts and regular bacteriological

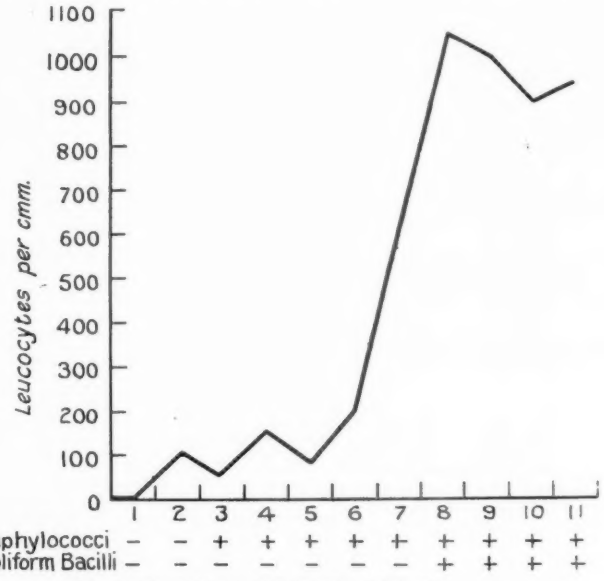


FIG. 4.—Incubation period of catheter infection.

examinations of the urine of these patients for a week before the operation and from four to six weeks after and have been able to trace the origin and course of their infections in an uninterrupted and leisurely fashion. A similar series of patients suffering from cancer of the colon, treated by a different operation not requiring the tying-in of a catheter, has provided me with controls of the same age and sex. I may dismiss these at once by saying that urinary sepsis does not regularly follow cancer of the bowel unless the patient is catheterized, and the urinary charts of these control cases never showed any rise in the leucocyte content of the urine after operation. The normal course of the infection which follows the tying-in of a catheter is as follows. The urine is sterile till the third day when staphylococci

first appear in cultures. During the first five days the urine may or may not contain a trace of pus, but from about the sixth to the eighth day the stream of pus steadily begins to flow, the urine-count rising from 100 to 1,000 cells per c.mm. in about forty-eight hours. About this time, if the case is destined to become complicated with a coliform bacillary infection, Gram-negative bacilli appear in company with the Gram-positive cocci on the surface of the nutrient agar. This punctual evolution is illustrated in the accompanying chart, representing the occurrences of the first ten days (fig. 4). The subsequent course of the infection cannot be foretold with the same precision. In most patients the sepsis lasts at least six weeks, and is accompanied by a continued pyuria with occasional remissions but no true intermissions. As the pyuria becomes less, coliform bacilli disappear from the cultures but staphylococci and sometimes streptococci persist for two or three days after the pus has disappeared. In the most favourable case so far observed, pus disappeared from the urine after twenty-seven days and the urine became sterile on the thirty-fourth day.

One of the most surprising features of this infection is the mildness of the constitutional disturbance. The continued daily excretion of urine containing more than 1,000 leucocytes per c.mm. is not reflected by any rise of temperature or pulse. It is not usually accompanied by any pain but the patient will probably complain of difficulty in holding urine, of difficulty in starting the act of micturition, a feeling of cramp, a desire to pass water frequently, and a continual sense of discomfort. This is attributed by the patient, and often by his attendants, to the severity of the operation of perineal excision of the rectum, but undoubtedly the true explanation is sepsis. In some cases the symptoms are more severe and interfere with sleep, but whether the symptoms are mild or serious, we cannot regard lightly any infection of the urinary passages accompanied by the growth of pathogenic bacteria and by the loss to the patient of something like a billion polymorphs a day, if his urine cell-count is 1,000 leucocytes per c.mm. and he is passing a litre of urine a day. I need hardly say that, now that I have extracted so much information from the study of catheter infections, steps have already been taken to avoid this troublesome and probably preventable complication of cancer of the bowel.

Section of Epidemiology and State Medicine.

President—Dr. S. Monckton Copeman, F.R.S.

[*March* 23, 1928.]

Season and Disease.

By C. O. Stallybrass, M.D., D.P.H.

Abstract.— The seasonal variations in disease, as is the case in the spread of infective diseases generally, depend upon three primary factors: (1) the presence of the micro-organisms of adequate virulence and infectivity; (2) the means of transmission to (3) the susceptible tissues of the susceptible individual. All other factors, in this relation largely climatic, can act directly only through these three primary factors and are therefore termed secondary. The paper is largely an attempt to express the effect of the variations in the secondary factors in terms of the alterations they produce in the primary factors. The total effect of the three primary factors in the spread of infection is termed "dispersability" and a ratio or measure of dispersability is described. This ratio emphasizes occurrences in the pre-epidemic period.

The relationship of temperature and humidity to the alimentary, respiratory and percutaneous diseases and to the ectodermoses, is investigated. In the autumnal group of infections, attention is drawn to the action of carriers in causing a rise in dispersability in the spring and the frequent occurrence of a double wave of dispersability.

The effect of climatic changes is often cumulative, and this is most evident in the autumnal group of infections, the seasonal occurrence of which cannot be directly explained by temperature changes, or even by the action of light on phagocytes. The possibility of vitamin deficiency or excess producing cumulative effects is considered.

Secular changes in seasonal periodicities are investigated and these are associated with other changes, such as the intrinsic periodicities, mode of spread, etc., the combined changes being described by the terms aggradation and degradation of disease.

Slides covering the majority of the infective diseases of temperate climates and exhibiting the secular changes in Liverpool, and various geographical differences, in some 175 curves displayed.

It is a coincidence that it is now almost exactly twenty years since Peters read his paper on "Season and Disease" before this Section [1]; he stated that the matter he then presented was only preliminary to a larger discussion of the question. Peters was struck down in Gallipoli, and his work was never completed. In his communication he dealt largely with two diseases, epidemic diarrhœa and enteric fever, and he investigated, with a wealth of detailed research, the relationship of climatic factors, more especially atmospheric temperature, to the rise of the seasonal wave and to its height in individual years in those two diseases. It is not my intention to follow this line so closely, but rather to deal with the subject on broader lines, such as the relationship to each other of prevalance and fatality, and also certain other statistical observations in a number of diseases.

An earlier generation of epidemiologists obtained their statistical data largely from death returns, and in some diseases we have still to rely on mortality statistics. Inferences were sometimes made from mortality back to morbidity, which were not always justifiable, for mortality is a compound of two variables, the product of prevalence and fatality. In fact mortality = prevalence × fatality.

Errors have also arisen, in the past, from an examination of the figures of an insufficiently lengthy series of years, thus causing failure to eliminate from the result irregularities due to transient causes, such as milk- or water-borne outbreak; and

also, for the same reason, the results will be affected by periodicities other than seasonal, i.e., the intrinsic periodicities which were so ably investigated by the late Dr. Brownlee, whose death we have had so recently to deplore. The larger the variations due to intrinsic periodicities, the more they will tend to mask the true annual cycle. Possibly some alterations in the annual cycle may result from alterations in the secular trend, as will be shown later.

The accumulated experience of nearly forty years of the notification of many infectious diseases in this country has provided a wealth of figures which cry for investigation and analysis, and very extensive series of figures have recently become available. By these means one is able to procure a smooth curve which is æsthetically satisfying; and for a long time, I must confess, I sought for such a series of figures, or such a manipulation of figures, as would give a smooth curve. Further thought has brought doubts into my mind—doubts which I hope to justify—as to the value of the smoothed curve. By extending one's series of cases either geographically or in length of years, is there not a tendency to lose the homogeneity of the series? The conditions of London and of a mining district in relation to enteric fever, for example, may be essentially different, and if the figures pertaining to a long series of years are placed together, secular changes which may be, and in fact are, in progress, may be masked. The ideal series of figures is, then, one which applies to a single town or uniform district and for several periods of about ten years, and I have investigated the figures relating to several diseases in Liverpool in this manner. I should have liked to extend this method of analysis to other places and other diseases, but the labour involved is considerable and time did not permit. In some diseases there is evidence to show that the figures over a number of years are homogeneous—undulant fever in Malta, for example—and there is no advantage in breaking them into decennial periods. In many diseases, however, I have to be satisfied with such figures as I can get. It is especially in relation to fatality rates that one is on the horns of a statistical dilemma, because in a disease with a low fatality-rate the influence of chance errors of sampling in a short series of years may be considerable. But if the same general curve recurs over a series of decades, one may be sure that one is dealing with a significant phenomenon. And this appears in relation to a considerable number of diseases (see diagrams VI, VIII, IX and XV).

In the seasonal variations of prevalence, as in the spread of infective diseases generally, three *primary* factors are in operation, which it is customary, by an analogy with the parable of the sower, to term, seed, sower and soil. These three factors may be more accurately defined:—

(*a*) *The seed.*—The reservoir of micro-organisms of adequate infectivity and virulence;

(*b*) *The sower.*—The adequate means of transmission to

(*c*) *The soil.*—The susceptible tissues of the susceptible individual(s).

In a paper which I read in April, 1922, on scarlet fever and diphtheria [2], I used the term "dispersability," which I coined for the purpose of expressing "the sum total effect of the three factors *a*, *b* and *c* in influencing the spread of a given infective disease among a given population at a given time and place." I was aware that Peters [3] had used the phrase "epidemic potential" to mean the balance between the infectivity of the causative organism and the susceptibility of the population; Peters' phrase does not appear to take into consideration the effect of factor (*b*), the transmitting agent which may—as, for example, in malaria—be of prime importance. Topley has recently extended the use of this phrase so as to have the meaning "balance of interacting forces which tends towards the occurrence of an outbreak of disease." Now, this definition seems to me to have almost the same meaning, though perhaps better expressed, that I intended to

give to my word "dispersability." I think that Peters' phrase has a definite value in its original meaning, so I propose to use the word dispersability in this paper. Dudley [4] has recently used the phrase "apparent infectivity" in this relation, but I consider this phrase unfortunate.

In the paper just referred to I said that the dispersability of a disease could be measured by its prevalence. This was a statement which I wish to modify. Prevalence at a specified time has an historic basis depending, as it does, upon what has gone before; at a given moment it is dependent upon the numbers of reservoirs of the virus existing at the moment, and also upon what one might perhaps term the intrinsic dispersability.

A morbidity rate is, therefore, a measure both of the intrinsic dispersability of the disease and of the numbers of foci of infection. Dudley [4] states that the "apparent infectivity" can be defined as "the number obtained when the attack rate is divided by the duration of the epidemic in days." This is, of course, useless for my present purpose, which is to estimate the dispersability of the disease, not throughout the whole epidemic, but at a given moment or period of time. This I propose to do by dividing the number of cases in period *b* (say February) by the number of cases in the preceding period *a* (say January) where *a* and *b* are successive and approximately equal periods of time. This ratio was used by Brownlee and Young in their paper on diarrhœa, read here in 1922 [8], and I propose to call it the ratio or "index of dispersability"—the difference between this ratio and a true index being so slight in most diseases as to be negligible. It has this significance that it represents the number of cases to which a single case in period *a* has, on the average, given rise in period *b*. I have introduced a curve of indices of dispersability into many of my diagrams. A word of caution must be given as to its statistical value. A rise from ten to fifteen cases in successive months will give an index identical with that given by a rise from 1,000 to 1,500, so that the index is more liable to errors of sampling during the off-season than during the season of maximum prevalence: this source of error must be discounted. But it has this advantage for my present purpose that it throws into relief those changes which take place in the inter-epidemic and pre-epidemic periods which, as Peters contended, were of the greatest interest.

At this point it will be questioned where, in the scheme just laid before you, is the influence of these climatic or other factors which control the seasonal variations of disease? The reply is that the climatic and all other environmental factors are merely *secondary* and indirect in this sense that they can affect the spread of the disease only by influencing one or more of the primary factors just defined. It is true that our knowledge of the exact manner in which these secondary environmental factors influence the primary factors is somewhat scanty; to express the relationship between the secondary and the primary factors will require much painful research, and this is one of the most promising fields before the epidemiologist. I will shortly give a brief résumé of our knowledge of this relationship as it affects seasonal variations of disease. But there appears no logical escape from the belief that, apart from the three primary factors, no other factor can *directly* influence the spread of disease; there is no room in this scheme for *quid mysteriosum* which can exert a direct action.

One more preliminary explanation which, though well known, is yet apt to be overlooked. Prevalence is dependent on the factors just mentioned; but death, and hence also a fatality-rate, is dependent in many diseases not so much on the primary invader as on one or more secondary invaders; in this climate deaths from measles are mainly deaths from pneumonia; but in Egypt, so I am informed by Dr. Hamdi Bey, enteritis is usually the fatal complication. Hence there may be,

not only the virulence of the primary virus and the susceptibility to death (shall we call it *mortability*) of the individual in relation to that virus, but also the same factors in relation to one or more secondary invaders; and these latter may have the more important bearing upon the chances of death. There is evidence to show that the main influence affecting seasonal changes of fatality in many diseases is the occurrence of respiratory complications. But not only does the fatality-rate of other

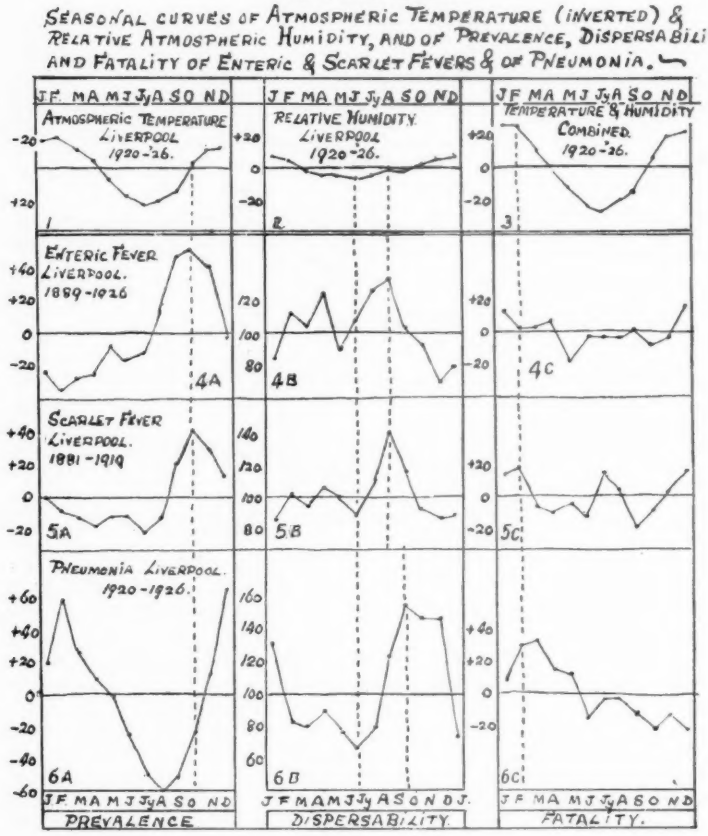


Diagram I.

diseases rise, up till about February, but there is a similar and well-marked rise from December onwards in the fatality of pneumonia itself, this rise reaching a well-defined maximum in February and March (see Diagram I). Eight of the commoner infective diseases of this country have their maximum fatality between December and April, of which five have their maximum fatality in February.

The climatic secondary factors which may influence the prevalence and fatality of infective diseases are, in themselves, sufficiently numerous; they are, (1) temperature, and (2) relative or absolute humidity of the atmosphere, (3) rainfall, (4) wind, (5) sunshine, (6) barometric pressure—although no *direct* influence attributable is known, and (7) possibly atmospheric electricity which Mohammed [5] has shown to be positively related to the occurrence of epileptic fits.

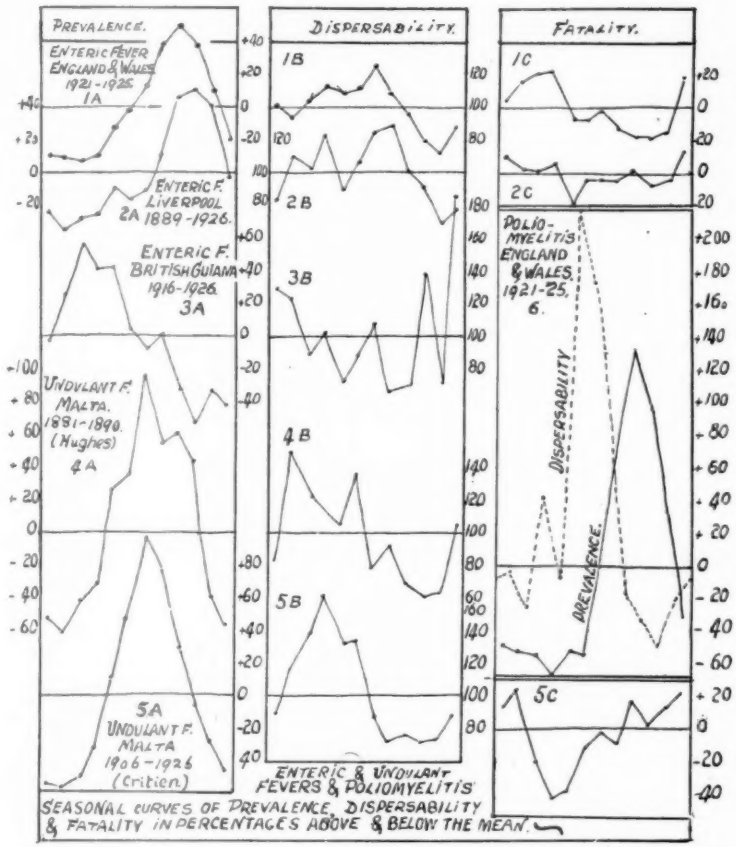


DIAGRAM II.

In addition there are influences which are not so much climatic as the effects of season upon human activities; school holidays, crowding in winter in trains, trams, or other meeting points, annual pilgrimages or festivals, such as move large bodies of people in India (and other Eastern countries) are examples.

Each of the secondary climatic factors in turn may possibly affect one or more of the three primary factors of seed, sower and soil, and may affect these differently

in each several disease. Moreover their action may be direct or indirect. Taking rainfall, for example, its action may be *direct* when heavy rain washes dejecta into a water-supply, *indirect* when an insufficient water supply dries up springs and compels recourse to a polluted supply; *indirect* when the temperature or rainfall permits or prevents the breeding of the mosquitoes that are the vectors of malaria, but *direct* when the temperature influences the viability of the parasite in the body of the mosquito.

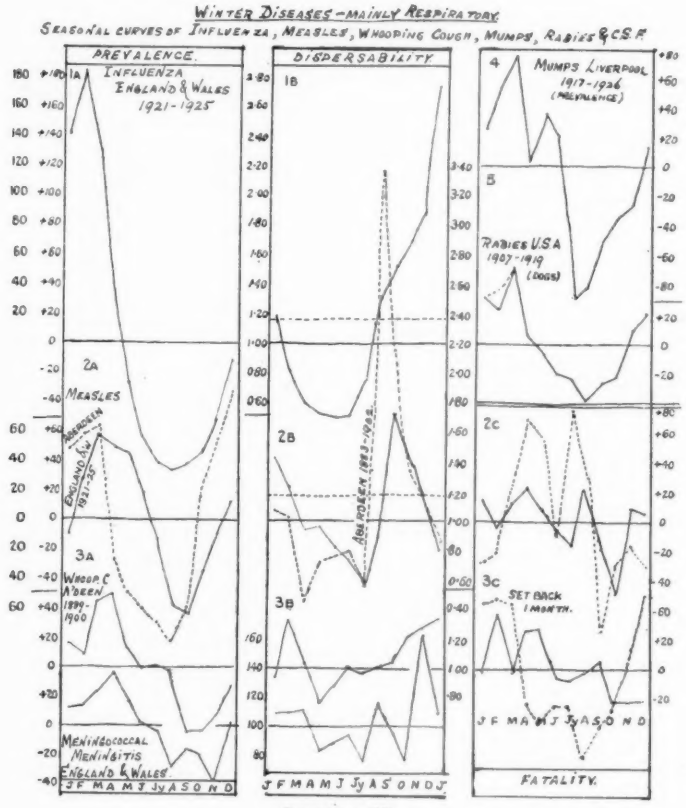

WINTER DISEASES—MAINLY RESPIRATORY.
SEASONAL CURVES OF INFLUENZA, MEASLES, WHOOPING COUGH, MUMPS, RABIES & C.S.F.
PREVALENCE.
DISPERSABILITY
1A INFLUENZA ENGLAND & WALES 1921-1925
1B
4 MUMPS LIVERPOOL 1917-1926 (PREVALENCE)
5 RABIES U.S.A 1907-1919 (DOGS)
2A MEASLES
ABERDEEN
ENGLAND & W. 1931-35
2B
ADELAIDE 1923-1928
2C
3A WHOOP.C ABERDEEN 1899-1900
3B
3C SET BACK 1 MONTH.
MENINGOCOCCAL MENINGITIS ENGLAND & WALES
J F M A M J Jy A S O N D
J F M A M J Jy A S O N D J
J F M A M J Jy A S O N D
FATALITY.

DIAGRAM III.

Each of the possible climatic factors may influence the spread of any given disease in one of several ways.

Broadly classifying infective diseases according to their portal of entry into alimentary, respiratory, and percutaneous infections, however, simplifies the investigation and we may proceed to do so.

(1) *Alimentary Infections* (see Diagram II).—It is forty years since Ballard [6] showed that in these climates there was a direct relationship between the temperature

recorded by the 4 ft. earth thermometer and diarrhœal mortality. This direct relationship of diarrhœa and temperature has been amply confirmed statistically in later years by the method of the coefficient of correlation. Thus Gibson [7] found that the coefficient between diarrhœal mortality during 1871 to 1913 and summer temperature (i.e., mean temperature of the third quarter of the year) had the high value of + 0·85 ± 0·04.

Brownlee and Young [8] found the coefficient between diarrhœa and temperature for the years 1856-1899 was + 0·80 and for the later years was + 0·75. These coefficients are highly significant.

(2) *Respiratory Diseases* (see Diagrams I and III), with which I group measles and whooping-cough, show an equally close relationship with low temperature; in other words their relationship to temperature is reciprocal. Moreover so marked is the influence of respiratory disease upon total mortality that the whole annual cycle of mortality closely follows the curve of temperature. Gibson [7] found that the coefficient of correlation for non-diarrhœal infant mortality and winter temperature was − 0·82 ± 0·05, again highly significant. This relationship is well demonstrated by the chart of mortality published by the Registrar-General in his Statistical Review for 1925. From this it will be seen that general mortality follows temperature with a lag period of about one month, respiratory disease, or respiratory complications in chronic diseases, being the predominant factor; other results such as the effect of cold in precipitating hæmorrhage in the arteriosclerotic or the phthisical also increase the mortality. Smiley [9] sums up the evidence in these words: "(1) There is apparently a definitely reciprocal relationship between the incidence of acute respiratory infections and the mean outside temperature. (2) There is apparently a definite reciprocal relationship between the incidence of acute respiratory infections and the average daily hours of sunshine." Capt. R. R. Evans, R.A.M.C., has recently calculated the coefficient of correlation between daily cases of influenza and atmospheric temperature in an outbreak at Woolwich and finds them highly correlated. Several of the ectodermoses such as smallpox, rabies and mumps, and, to a less extent, chickenpox, also exhibit this inverse relationship of prevalence and temperature. These all have a seasonal range varying from about 50 per cent. below the mean to 50 per cent. to 80 per cent. above the mean. The similarity of the curves is remarkable when we consider the differences in the mode of transmission of small-pox, rabies and mumps.

(3) *Percutaneous Diseases.*—These, in so far as the insect-borne diseases of temperate climates are concerned, show a summer maximum for those conveyed by flying insects and a winter maximum for those conveyed by lice. (See Diagram IV, 2 and 3.)

Broadly speaking, then, in temperate climates there is a direct relationship between temperature and prevalence in alimentary infections and those conveyed by flying insects on the one hand; and a reciprocal relationship between temperature and respiratory diseases, a number of the ectodermoses, and louse-borne infections, on the other. (See Diagrams III [4 and 5], and IV.) But a closer study shows that the relationship is not so simple as would appear from this statement; there is in most infective diseases a lag period between the maxima—or minima—of temperature and the maximal prevalence of disease, and this lag period shows considerable variations between one disease and another depending, doubtless, upon the exact manner of spread of different diseases, upon their incubation periods, and possibly upon other factors. Moreover, to my mind, what is of importance is the rise of dispersability which usually reaches its maximum some months before prevalence does so. The effects of temperature changes, in fact, are not only immediate but cumulative. As Peters pointed out, in relation to diarrhœa and

enteric fever, the seasonal variations depend, not so much upon a mere arrival at a certain critical temperature, as upon the maintenance of that temperature for a certain length of time; so that the point which marks the *main* rise of these disease marks also the end of a period of several weeks' duration during which the influences of certain summer temperatures were in evidence. This he termed the

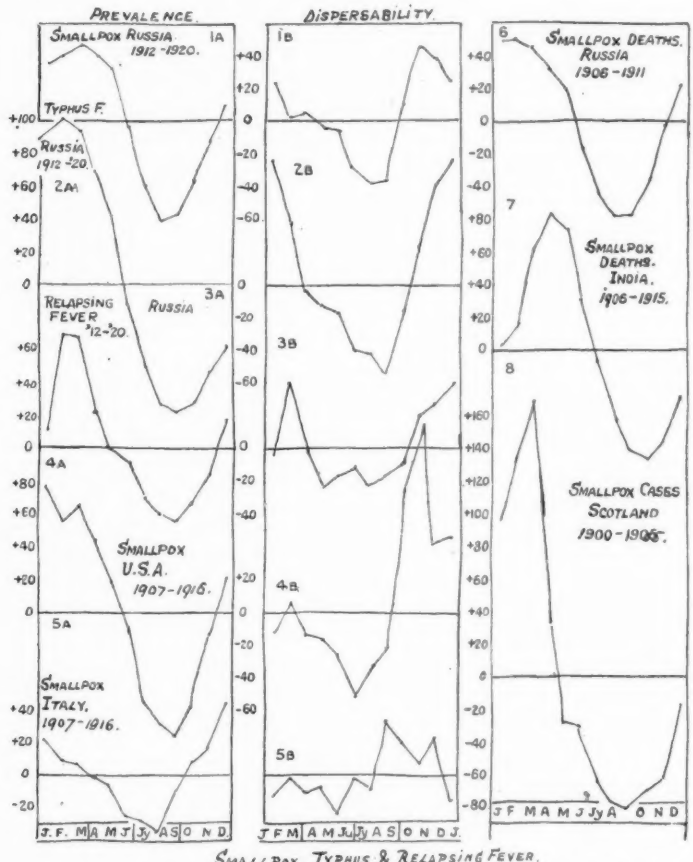


DIAGRAM IV.

pre-epidemic period. I would point out that the index of dispersability attracts attention to changes taking place in the pre-epidemic and inter-epidemic periods.

Further, the relationship between temperature and prevalence in a given disease is not always constant but in certain diseases shows variations of a secular character both in time and in range of variation. I will consider this later.

Moreover, there is a considerable group of diseases which is mainly autumnal in incidence and has a doubtful association with the temperature curve. This group (see Diagrams V and VII) includes the throat infections such as diphtheria and Vincent's angina and a considerable group of streptococcal infections amongst which we may place scarlet fever. Enteric fever, which would naturally fall among the

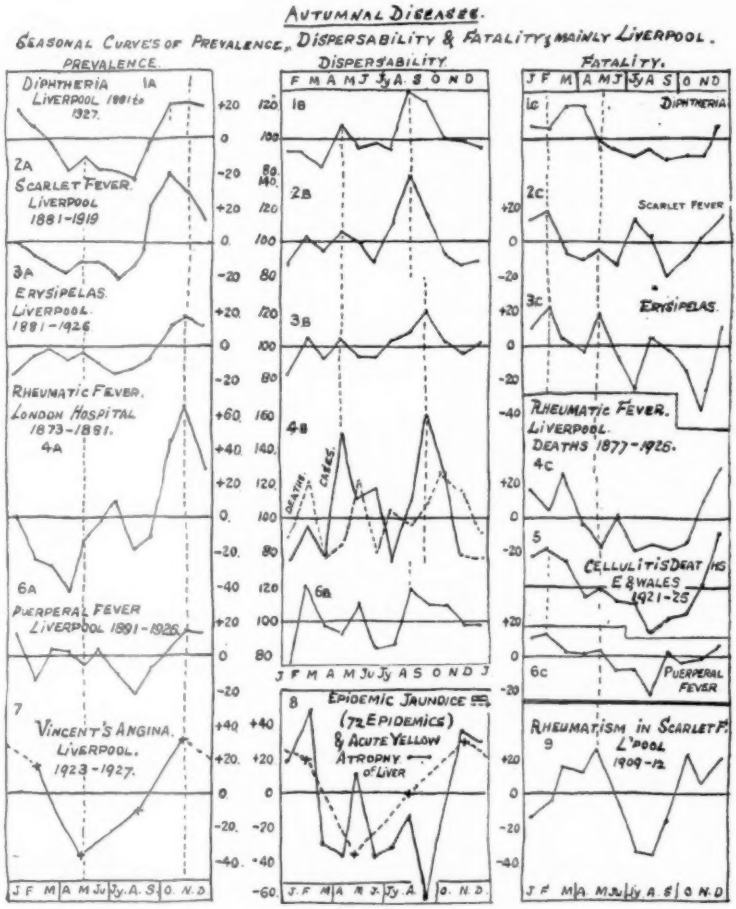

AUTUMNAL DISEASES.
SEASONAL CURVES OF PREVALENCE, DISPERSABILITY & FATALITY; MAINLY LIVERPOOL.
PREVALENCE.
DISPERSABILITY.
FATALITY.
DIPHTHERIA 1A LIVERPOOL 1881 to 1927.
SCARLET FEVER. LIVERPOOL 1881-1919
ERYSIPELAS. LIVERPOOL. 1881-1926
RHEUMATIC FEVER. LONDON HOSPITAL 1873-1891.
PUERPERAL FEVER LIVERPOOL 1891-1926
VINCENT'S ANGINA. LIVERPOOL. 1923-1927.
DEATHS & CASES.
EPIDEMIC JAUNDICE (72 EPIDEMICS) & ACUTE YELLOW ATROPHY of LIVER
RHEUMATIC FEVER. LIVERPOOL. DEATHS 1877-1926.
CELLULITIS DEATHS E & WALES 1921-25
RHEUMATISM IN SCARLET F. L'POOL 1909-12
DIPHTHERIA
SCARLET FEVER
ERYSIPELAS
PUERPERAL FEVER
J F M A M Ju Jy A S O N D

DIAGRAM V.

summer alimentary infections, now often shows this autumnal activity. It has been previously shown that it has the same relationship to temperature as the other alimentary infections, namely cholera, dysentery and epidemic diarrhœa, but that it has a long lag period. I am able to confirm this relationship in a new way. Some years ago I worked out for a period of sixteen years in Liverpool, a curve of

coefficients of correlation between the monthly number of cases of enteric fever and the mean temperature for that month and the preceding two months (see Diagram XI). This showed that for the months of the annually recurring epidemic, namely September and October, there was a high correlation between these two sets of figures, giving for September a coefficient of + 0·945 ± 0·018 but that for the remainder of the year the correlation was effectively *nil*.

In the case of enteric fever, then, we have a cumulative effect of atmospheric temperature prolonged even for months after the maximum temperature has been reached. Why should enteric differ from the other three alimentary infections in

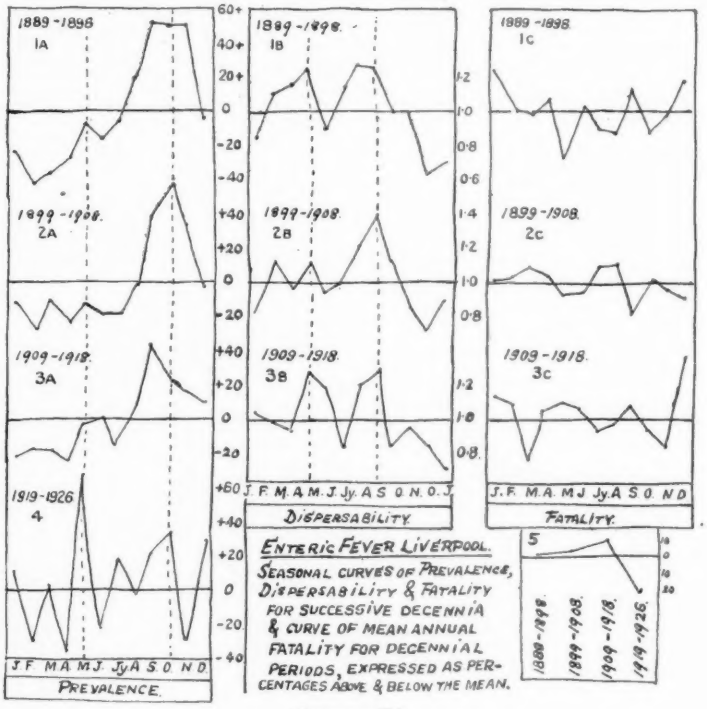


DIAGRAM VI.

this respect? One explanation leaps to the mind. It is that cholera, dysentery and diarrhœa have very short incubation periods, but that enteric fever is long in developing; so that, other things being equal, in this latter disease the number of foci of infection are slow in multiplying but that the explosiveness of the former three diseases is rather due to the shortness of the incubation period and the rapid multiplication of their foci. An examination of the curves of dispersability of these diseases shows (see Diagram XI), however, that whilst cholera, dysentery and diarrhœa exhibited single curves with maxima in May-June or June-July, enteric fever, on the other hand, has a double curve of dispersability with maxima in April-

May and August-September. Investigation week by week of the mortality from these three diseases in Liverpool during the fifties of last century showed that the maximal dispersabilities were in the twenty-fourth, twenty-fifth and twenty-eighth weeks for cholera, diarrhœa and dysentery respectively; and the corresponding maximal prevalences were in the thirty-third, thirty-fourth and thirty-fifth weeks, i.e., about seven to nine weeks later. The difference between enteric and this triad

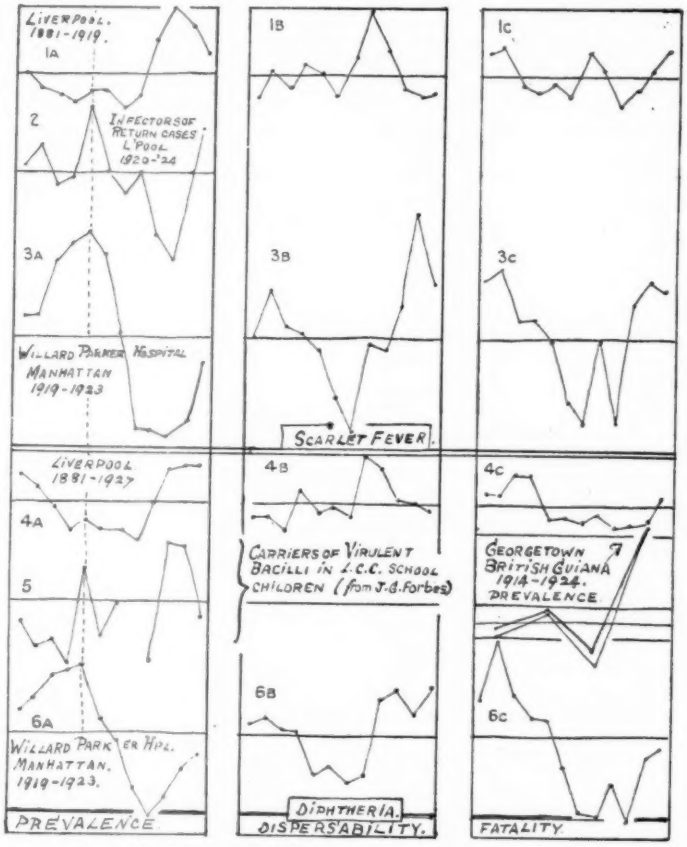


DIAGRAM VII.—Scarlet fever and diphtheria. Comparison of seasonal curves of prevalence in Liverpool, New York and Guiana, and of the occurrence of carriers.

of intestinal diseases is perhaps something more fundamental than the mere length of their incubation periods.

This definite rise of enterica in May has been noted by other observers—it is shown in a diagram of Mair's relating to Belfast, 1897-1906 [10], and in the Registrar-General's figures, relating to England and Wales for 1921-1925, but its

significance has, I think, been possibly overlooked. Some years ago I noted that two chronic typhoid carriers most often gave rise to other cases in their family in the spring of the year, namely, about May. As the cases were so limited I did not consider it worth while to draw attention to it, but as the observation fits in with others I now do so, and should like to have it confirmed or corrected in relation to other chronic typhoid carriers.

Returning to the autumnal group of disease, the throat and streptococcal infections, it is obviously impossible to account for their prevalence in autumn in relation to the length of the incubation period. An investigation of the curves of dispersability (see Diagrams V to IX) shows that they all present a double or dromedary curve with maxima in April-May or May-June and August-September or September-October. Moreover, two of them, namely, scarlet fever and diphtheria, exhibit another feature, namely, that carriers appear to show increased activity in the spring, more especially about May. The curve of carriers of virulent diphtheria among London school children in 1921 and 1922, for which I have to thank Dr. J. Graham Forbes,[1] shows a double type with maxima in May and October (see Diagram VII, 5). My own curve of infectors of return cases of scarlet fever shows a well-marked maximum in May, whilst that of the Metropolitan Asylums Board [11] shows a somewhat earlier maximum, namely, February and March; it is customary to attribute this increased activity of the carriers to the prevalence of catarrhal complaints or colds which increase the infectiveness of the carriers, but there may be other factors. It is interesting to note that in New York, and, I believe, Paris too, both scarlet fever and diphtheria have recently been spring and not autumn diseases; in New York the maximal prevalence for both diseases was in May although the maximal dispersability was in the late autumn. I am unable to suggest what the explanation of this inversion of prevalence may be.

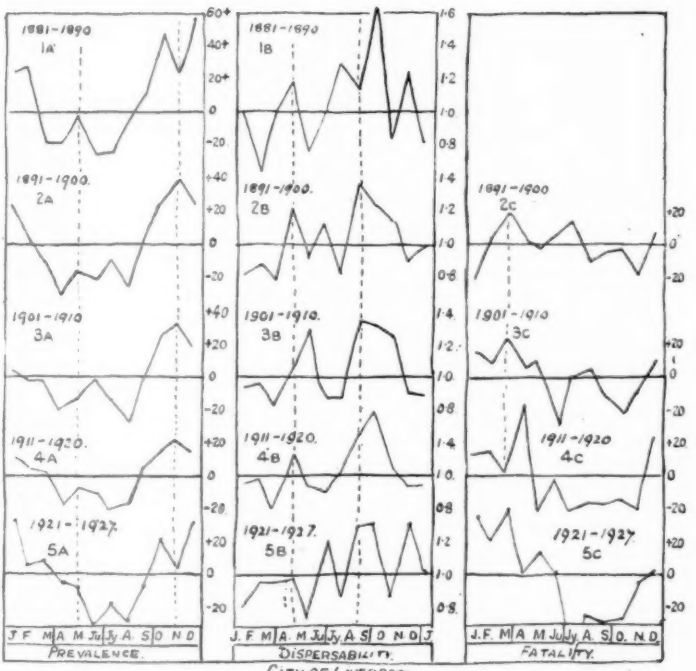
A further line of investigation of the relation of temperature and prevalence suggests itself; there are places in the tropics, such as British Guiana and Singapore, where the climate is remarkably equable; if the temperature were the main factor then they should exhibit little seasonal fluctuation. Figures relating to British Guiana, kindly obtained for me by Dr. Haslam, show that in that country enteric fever and diphtheria have seasonal curves closely corresponding in range of variation with those of the British Isles. But whilst diphtheria is there, as here, an autumnal disease (see Diagram VII) enteric fever is a vernal disease, with a maximum in March (see Diagram II). There appear to be two wet and two dry seasons in Guiana, the dry seasons being February to April and September-October, though these are uncertain; but these are relative terms, the rainfall seldom falling below 6 in. in any quarter of the year, although occasionally there are prolonged droughts.

The figures that I have for Singapore are inadequate, as they only relate to the deaths in the three years 1923-1925. They indicate moderate seasonal variations for mortality in dysentery and enteric fever; the mortality from dysentery reaching a maximum in June, July, or August, of 13 per cent. above the mean, and that from enteric fever (based on only 174 deaths) having two maxima, one in August and one in November-December.

What are we to make of these contradictions? It is clear that no simple correspondence between temperature and prevalence or dispersability will fit the case of more than a very few diseases, even when we allow for considerable lag periods. It remains to find some other factor which alone, or in combination with temperature changes, gives a high correlation with prevalence or, as Brownlee did in the case of

[1] Personal communication.

diarrhœa, with the dispersability ratio. But correlation with two or more factors can only be carried out exactly by the method of partial and multiple correlation, and this is, as Brownlee termed it, "a labyrinth without a clue." Recently Major Russell, associated with the Sundararajan [12], has investigated the somewhat bewildering contradictions presented by the variations of seasonal prevalence of cholera in India. I do not propose to lead you into that maze; suffice it to say that their conclusions were that, allowing for the high correlation of rainfall with both temperature and humidity, "the net association of rainfall with cholera is distinctly less than was indicated by the single correlation." Further, "when temperature



CITY OF LIVERPOOL.

CURVES OF PREVALENCE & FATALITY – IN PERCENTAGES ABOVE & BELOW THE MEAN – & OF INDICES OF DISPERSABILITY OF DIPHTHERIA IN SUCCESSIVE DECENNIA.

DIAGRAM VIII.

effects are ignored, the association of humidity with cholera is highly significant." Sir Leonard Rogers [13], [14] has indicated that there is an inverse relationship between smallpox prevalence and absolute humidity of the atmosphere both in India and in Great Britain.

Even when in reference to a given disease, or group of diseases, such a relationship between some climatic factor or factors and the prevalence or dispersability has been established, there remains the much more important step of discovering in what manner these climatic factors influence the primary factors of "seed, sower and soil," and upon which primary factors they operate. This knowledge in the past

has been obtained mainly in relation to the activity of the transmitting agent. High temperature and low relative humidity destroy the plague bacillus sprayed out from cases of pneumonic plague, and probably affect most droplet-borne respiratory diseases in a like manner, but a lesser degree. High temperature and high relative humidity permit the breeding of flies; but high temperature and low relative humidity stop their pullulation. Observations upon diarrhœa indicate a relationship variable, but positive, between fly prevalence and diarrhœal mortality, which in this country both present single curves; but in Macedonia [35], owing to the low summer

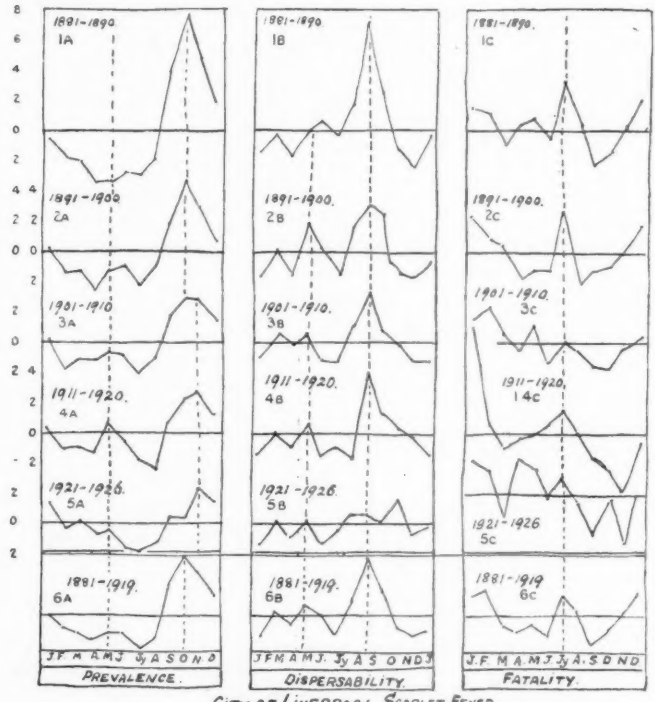


DIAGRAM IX.

rainfall the relative humidity is too low in summer to permit of flies breeding copiously, and so we get a double verno-autumnal curve of fly prevalence and a like double curve of dysentery prevalence. Why does not someone investigate fly-prevalence in India in relation to cholera, if this has not been done? The combined effects of temperature and relative humidity go far to explain the seasonal variations of both respiratory and alimentary infections.

But what of the influence of climatic factors upon the two other primary factors in disease prevalence—the seed and the soil? Here we are upon much more uncertain ground. As to seasonal variations in the virulence or infectivity of the virus, though

there have been many speculations, I know of only one direct observation. Powell, Atwater and Felton [15] obtained what they have termed "a virulence index" in relation to pneumococci. The virulent strains of pneumococci are most frequently found in association with cases of pneumonia and there is a suggestion that the virulence index shows seasonal fluctuations, with a maximum virulence in December. Gaskell [16] has confirmed these results and has shown that for three types of pneumococci there is a definite decline in virulence in the summer with a corresponding rise in the winter. Taking into consideration the naturally variable virulence of pneumococci and the considerable range of virulence actually demonstrated, we may consider that they have excluded the effects of reciprocal variations in the susceptibility of the mice; otherwise it is, indeed, difficult to see how this can be done. As I have already stated, the fatality rate of pneumonia rapidly rises between December and March, and falls almost as rapidly between March and June. This is in marked relationship, though with a lag of one month, to the atmospheric temperature, but whether it is mainly an effect of increased virulence or of increased susceptibility must remain in doubt. It will be obvious that in investigating variations in the infectivity of the virus, it is variations in the dispersability of the disease and not its prevalence that are important.

There is a very attractive hypothesis that has been put forward by Topley that virulence and infectivity are reciprocally related, and I have endeavoured to find if there is any support for this in relation to seasonal fluctuations of prevalence and fatality. At first sight I found evidence to support this thesis in relation to measles and scarlet fever based upon smoothed curves. I am now of the opinion that the main seasonal variations in fatality depend upon the more frequent occurrence of respiratory complications in winter as almost all diseases show a maximum fatality in winter. In several diseases, however, there is evidence of a sudden rise in fatality accompanying the first seasonal rise of prevalence, i.e., the main rise in the dispersability curve; this is well shown in relation to scarlet fever (see Diagram VII, 1a and b). But as the seasonal epidemic progresses the curves of dispersability and fatality part company and go opposite ways. In one disease in which we are able to exclude the influence of pneumonia as a complication, namely in pneumonia itself, the maximum dispersability is reached in September-October and the maximum fatality in March, so that the inverse relationship postulated by Topley does hold good in this disease. It would be of interest to know the variations of fatality in measles in, say, Egypt, but I fear the figures are not available.

As to the influence of climatic changes upon susceptibility, there is much more direct evidence. Pasteur's observations upon the influence of external temperature upon the susceptibility of fowls and frogs to anthrax are classical, and Strouse has recently shown that a temporary reduction of body temperature renders pigeons susceptible to infection with pneumococci, to which infection they are normally immune. This evidence is entirely in accord with the prevalence of respiratory infections. Findlay [17] and Werkman [18] with birds, and Findlay and Maclean [19] with rats, have found that increased susceptibility to infection is associated with persistent lowering of body temperature caused by diets deficient in vitamin B. Whilst this demonstrates the effect of lowering of body temperature, I do not know of any grounds for believing that we, in this climate, suffer any seasonal deficiency of vitamin B in our dietary, nor that our body temperature is lower in winter than in summer. Moreover, Kon and Drummond [20] refer the lowering of body temperature to partial starvation and not to deficiency of vitamin B.

In relation to summer diarrhœa, Professor Ylppö [21] has recent made interesting observations. He found that exposure of young children to the direct heat of the sun is associated with a rise of temperature, which may reach 40·5° C. This

pyrexia produces a relative hypochlorhydria, and the infants exposed to the sun suffered from diarrhœa and vomiting. In experimentally induced transient fever, he was able to show that the acidity of the gastric juice fell from a figure of about 4·0, which readily destroys typhoid and dysentery organisms, to a value of 5·6 or 5·8, at which level the acidity of the gastric juice exercises a negligible bactericidal influence upon those bacteria. A word of caution is needed, as the bactericidal influence of the gastric juice may not be entirely dependent upon its acidity; but these experiments appear to afford a basis for a unifying theory in relation to the production of zymotic diarrhœa, according to which there is both increased susceptibility to infection during hot weather, as well as increased liability to be infected. Possibly also this observation of Ylppö may have some bearing upon the observations of Hunter, that pernicious anæmia almost invariably commences in the summer months.

About the effects of humidity upon susceptibility we know rather less. The effect of rain-bearing winds upon the prevalence of tuberculosis has been emphasized by Gordon in Devonshire, and Rogers [22] has laid the same stress upon the effects of the monsoons upon the prevalence of leprosy, as well as of tuberculosis, in India. An asthmatic is often almost as sensitive to changes of humidity as a hygroscope. But concerning the exact manner of action of humidity on human susceptibility to infection we have little knowledge.

The effects of temperature upon human susceptibility would appear to be temporary in their nature. The rapid rise of respiratory mortality consequent upon a cold spell soon passes away when more normal climatic conditions are regained. Are there then no effects of climate upon susceptibility which are cumulative in effect because a cumulative effect is necessary to explain the lag of some diseases behind the climatic changes? There are, of course, other explanations of this lag such as the interval between infection and manifestation of illness or occurrence of death, the persistence of infection in a community after the circumstances which, in the first instance, produced the spread of infection, and so forth; but may there not be a cause of human susceptibility which is cumulative? The discovery of vitamins which are dependent upon the actions of sunlight, upon plants or animals, has given us knowledge of a climatic agent, namely defect of sunshine or of ultra-violet light, which is possibly capable of producing this cumulative effect. I have placed together (in Diagram X) a number of curves associated with these seasonal nutritional disturbances. The curve for rickets is based on the date of falling sick with the first symptoms of that disease; that for scurvy in Russia, based on a vastly greater number of cases, is not strictly comparable, as it expresses the months of reporting of the developed disease. Both these curves may be compared with those of respiratory diseases, including measles and whooping-cough, and also with the curve of putting on weight in sanatorium; this latter is almost the reverse of the scurvy curve and shows that tuberculous subjects gain in weight most rapidly in the autumn, just as do growing children. I have also placed alongside these curves those of tetany (from figures given to me by Dr. Edmund Hughes [23, 24] and of serum-rashes in diphtheria (Brownlee [25]); these closely correspond to each other and also to the figures given by Hess as to the calcium and phosphorus content of the blood in infants which varies from a maximum in June or July to a minimum in March. Cannon finds the iodine content of the thyroid gland to be greatest in September or October and least in March or April, an observation bearing upon the fourfold rapidity of growth in children in autumn compared with the spring. Dr. W. E. Cooke informs me that thyroid extract administered to rabbits stimulates the production of polymorph leucocytes.

There is some evidence that vitamin deficiency does affect resistance to disease. Findlay [17, 26] has found that guinea-pigs suffering from scurvy show a lowered resistance to bacterial infections, and reference has already been made to the effects of deficiency of vitamin B in reducing body temperature. But apart from this, there are direct effects of sunlight upon resistance to infection. Colebrook, Eidinow, and Leonard Hill [27] found that exposure to ultra-violet light increased the phagocytic activity of the leucocytes, and Colebrook [28] confirmed this in relation

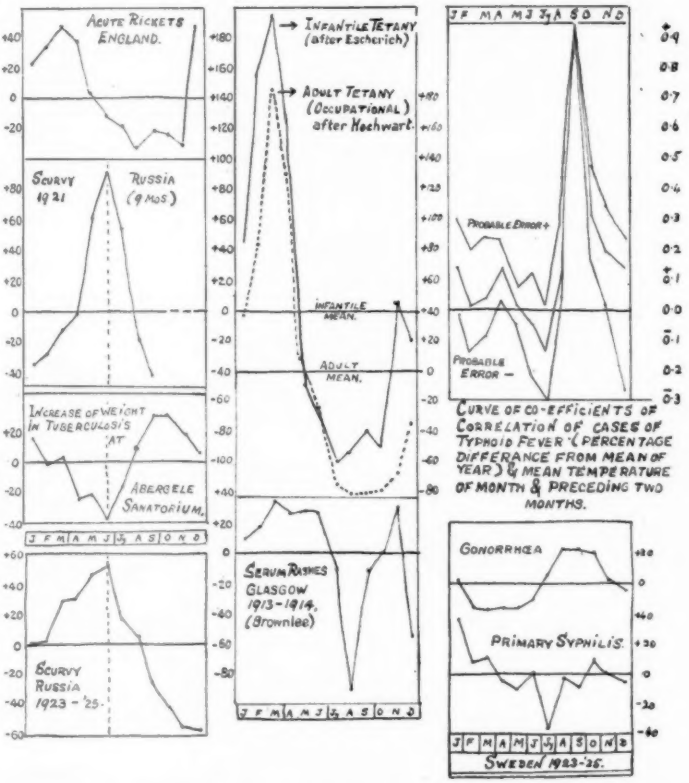
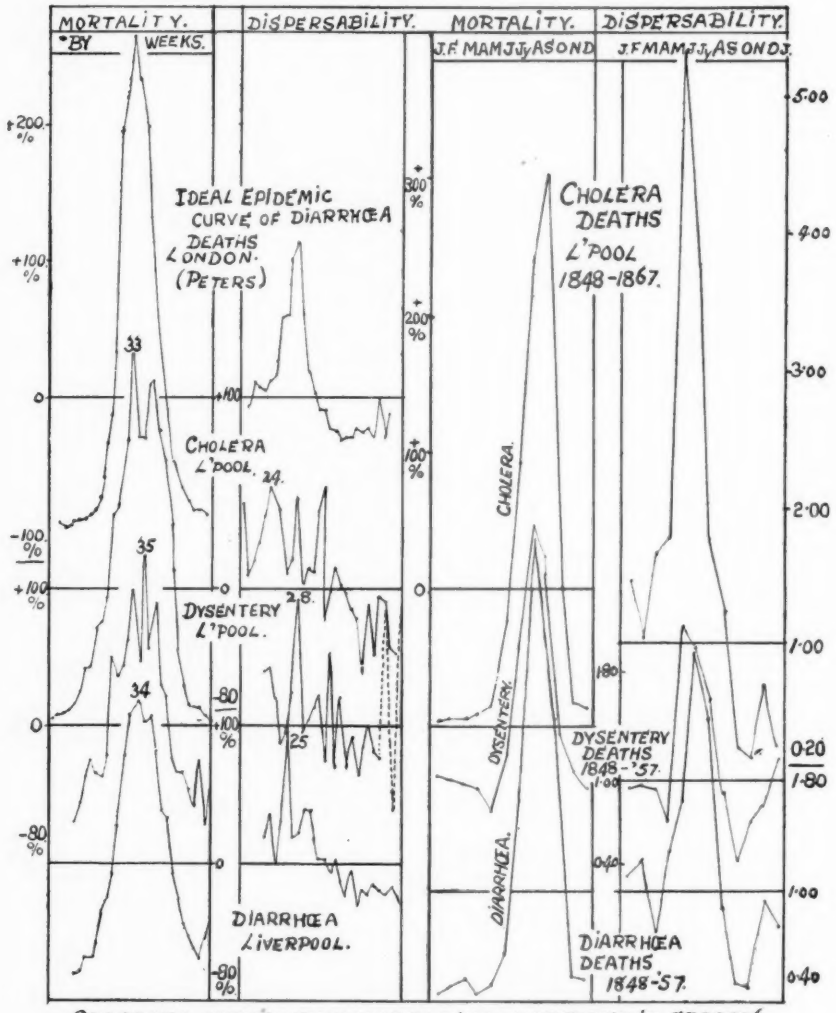

Acute Rickets England.
Scurvy 1921
Russia (9 mos.)
Increase of weight in Tuberculosis at
Aberdele Sanatorium.
Scurvy Russia 1923 - '25.
Infantile Tetany (after Escherich)
Adult Tetany (Occupational) after Hochwart.
Infantile mean.
Adult mean.
Serum Rashes Glasgow 1913-1914. (Brownlee)
Probable Error +
Probable Error −
Curve of co-efficients of correlation of cases of Typhoid Fever (percentage difference from mean of year) & mean temperature of month & preceding two months.
Gonorrhœa
Primary Syphilis.
Sweden 1923-'26.

DIAGRAM X.—Seasonal curves of avitaminoses and allied conditions and of venereal diseases. Also a seasonal curve of coefficients of correlation of temperature and prevalence of typhoid fever.

to sunlight. Werkman [18] had already shown that deficiency in vitamins A and B depressed phagocytic activity without, however, affecting the capacity to produce opsonins or other antibodies. Irala [29], however, found that over-exposure to ultra-violet light eventually injured the leucocytes and arrested phagocytosis. The effects of sunlight in precipitating pellagra is well known, and this disease shows a seasonal increase in the United States which is earlier in onset



SEASONAL CURVES BY WEEKS DURING THE EPIDEMIC SEASON, AND BY MONTHS FOR THE ANNUAL CYCLE OF CHOLERA, DYSENTERY & DIARRHŒA IN LIVERPOOL. ALSO A SUPERPOSED CURVE OF DIARRHŒAL MORTALITY IN LONDON. MODAL WEEKS ARE GIVEN IN FIGURES ABOVE THE CURVE TO WHICH THEY RELATE.

DIAGRAM XI.

the more southerly the State. Nevertheless, Claire Hill and Janet Clarke [30] say that "the present state of our knowledge concerning the effect of ultra-violet radiation gives very little support to the belief that it is capable of increasing natural resistance in normal individuals."

But if deficiency of vitamins can reduce the resistance of the host to infection why should there not be an action of vitamins in exalting the activity of the virus? The French biochemists believe that there is a vitamin, which they term D, which is stimulant to the growth of micro-organisms (Funk [31]) and it is at least conceivable that an increase during the late summer of the vitamins contained in the bodies of their hosts would increase the activity of the virus; this might serve for an explanation of the autumnal diseases which show a cumulative association with atmospheric temperature.

I now come to the somewhat vexed point of the causes of secular alteration—usually retardation—in the seasonal curve. This point was discussed in relation to summer diarrhœa by Brownlee and Young in the paper which they read in 1922 [8]; they very ably put forward the view that diarrhœal mortality represented not one disease but two and that these had seasonal maxima separated by an interval of six to seven weeks; further, that as the type with the earlier or summer incidence represented a more easily eradicable disease, the later, or autumnal, type has become more prominent, "by a process," as Sir George Newman has said in another relation, "resembling that of geological denudation."

I do not wish entirely to dissent from this view; undoubtedly dysenteric infections were formerly much more prevalent than they now are. But apart from dysentery I know of no clinical grounds for separation of diarrhœa into two diseases, and so I would apply to Brownlee's thesis William of Occam's razor: *entia non sunt multiplicanda praeter necessitatem.* Indeed, I have curves here (Diagram XI) which show that in the decade 1848-57, when the three diseases, cholera, dysentery and diarrhœa, were prevalent in Liverpool, they all reached their maxima within two weeks of each other. Further, the curve for cholera, as well as that for diarrhœal mortality, shows a dromedary or bifid apex, a feature not caused artificially by addition of two epidemics but recurring in each of the main epidemics that occurred in 1849 and 1854; this bifid apex is also shown, though less characteristically, by the curve of dysenteric mortality. The curves (shown in Diagram XI) all exhibit a close resemblance to Peters' summation curve for diarrhœa in London (produced by placing the peak mortalities under each other and adding together all the first, the second, and so on, weeks before and after the peak; in this way an ideal curve is produced which eliminates chance fluctuations due to the climatic vagaries of individual years).

The retardation of the apex of mortality is not confined to zymotic diarrhœa. Other diseases, notably typhoid fever and scarlet fever, show similar features. Indeed not only is there a retardation of the peak week, but there is a general flattening of the curve, and also a greater scatter of the modal weeks or months when a chart of their frequency distribution is prepared (see Diagrams XII and XIV). The chart for diarrhœa shows that the modal week has been retarded from the thirty-third to the thirty-eighth week in the course of eighty years; this retardation has not, however, been continuous, but during the last decade of last century and the first decade of this century there was an anticipation of the peak, and in 1898 to 1907 the modal week was the thirtieth. In enteric fever, formerly an August or September disease, there has also been retardation and increased scatter so that in Liverpool during 1921 to 1927 there has been hardly any characteristic seasonal curve; the peak in May, 1925, was due to an outbreak of paratyphoid B of unknown causation. In both diseases there are, or have been, differences between

the seasonal curve in one part of the city and another. Typhoid fever in 1911, for example, as I showed in a paper read in 1912 [32] was still markedly epidemic in the central insanitary areas of the city but showed much less seasonal variation—apart from cases due to consumption of infected shellfish, etc.—in the rest of the city. The decline of typhoid fever in Liverpool coincided with remarkable closeness with the destruction of insanitary areas during the years 1896 to 1911, the coefficient

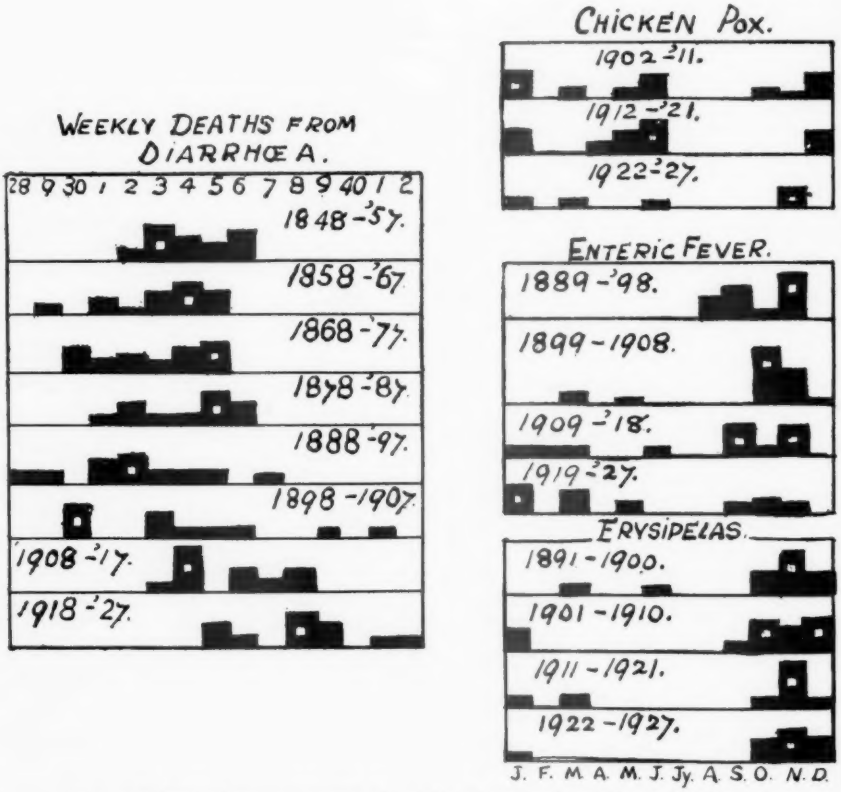


DIAGRAM XII.—Frequency arrays of months of maximal prevalence of chicken pox, enteric fever and erysipelas, and of weeks of maximal mortality of diarrhœa, by successive decennia in Liverpool. The modal months or weeks are indicated by a white spot.

of correlation between annual cases of typhoid and annual numbers of insanitary courts during those years reaching the highly significant figure of $+ 0{\cdot}9356 \pm 0{\cdot}002$.

In 1926 and 1927 diarrhœa was epidemic in the same Central area as was typhoid in 1912, and reached its maximum earlier there than it did in other parts of the city [33]. In both cases I am inclined to attribute the influence of the seasonal

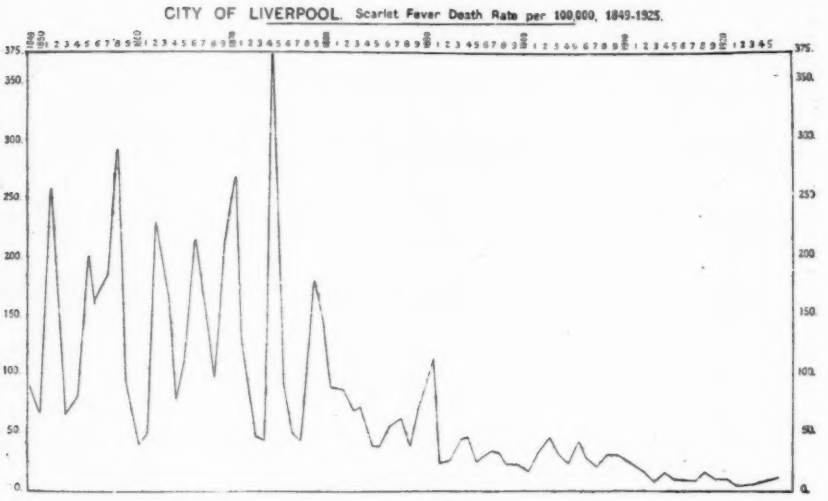


DIAGRAM XIII.

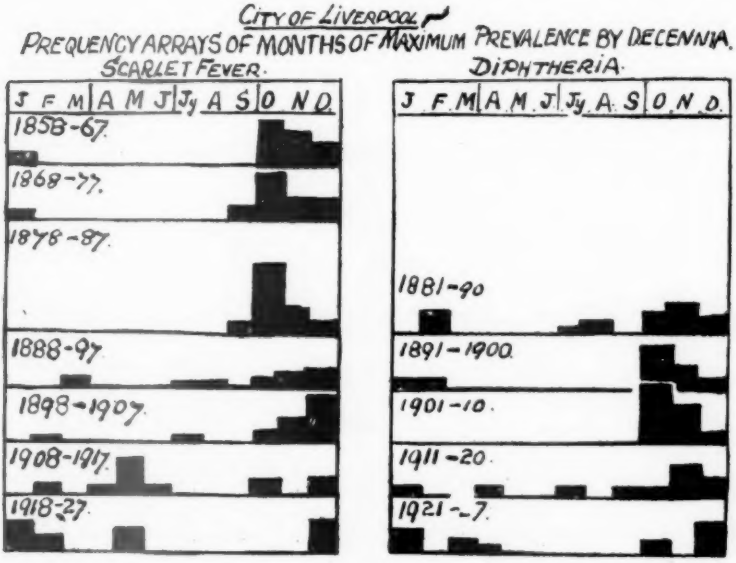


DIAGRAM XIV.

TABLE SHOWING THE SEASONAL MAXIMA AND MINIMA IN RELATION TO PREVALENCE, DISPERSABILITY AND FATALITY FOR 34 DISEASES OCCURRING IN TEMPERATE CLIMATES. THE FIGURES SHOW THE INDEX NUMBERS, THE MEAN BEING 100 FOR PREVALENCE AND FATALITY, AND 1·00 FOR DISPERSABILITY.

Diseases	Locality and Years	No. of Cases (c) or Deaths (d)	Indices of Prevalence Maximum	Prevalence Minimum	Dispersability † Maximum	Dispersability † Minimum	Fatality Maximum	Fatality Minimum
I.—Ectodermoses.								
Smallpox	Scotland, 1900-05	6,658 c.	March (170)	August (23)	Dec. (2·36)	March (0·50)	—	—
,,	U.S.A. [36], 1907-16	100,615 c.	January (177)	September (24)	Oct. (2·16)	June (0·50)	—	—
,,	Italy [36], 1907-16	49,650 c.	December (144)	August (65)	Aug. (1·34)	May (0·76)	—	—
,,	European Russia [36], 1906-11, 1912-20	224,589 d.	February (151)	September (42)	Oct. (1·53)	July (0·65)	—	—
		825,807 c.	March (148)	August (39)	Oct. (1·47)	July (0·63)	—	—
,,	India, 1906-15	945,674 d.	April (183)	October (33)	—	—	—	—
Varicella	Liverpool, 1902-06 and 1912-20	29,920 c.	June (153)	August (58)	Oct. (1·23)	August (0·72)	—	—
Mumps	Liverpool, 1917-26	17,240 c.	March (176)	July (12)	Sept. (2·99)	[July 0·09]	—	—
Rabies	Washington, U. S. A. [38], 1909-19	588 dogs	March (153)	August (61)	Nov. (1·42)	March (0·69)	—	—
Encephalitis lethargica	England and Wales [39], 1921-25	— c.	May (196)	November (56)	Jan. (1·66)	May & June (0·63)	—	—
Poliomyelitis	England and Wales [39], 1921-25	1,916 c.	September (233)	April (35)	July (2·17)	November (0·51)	—	—
,,	Sweden [40], 1911	3,840 c.	September (333)	January (1)	April (3·66)	December (0·46)	—	—
II.—Respiratory Infections.								
Measles	Aberdeen [41], 1883-1902	40,374 c.	March (163)	August (18)	Aug. (3·36)	March (0·45)	April (169)	Sept. (28)
,,	England and Wales [39], 1921-25	— c.	March (158)	September (37)	Nov. (1·73)	September (0·47)	April (124)	Oct. (51)
,,	Manhattan [42], 1919-23	3,720 c.	May (186)	October (14)	Nov. (2·02)	August (0·50)	Jan. (144)	Sept. (0)
Whooping-cough	Aberdeen [43], 1883-1902	16,723 c.	April (150)	September (54)	Jan. (1·34) March (1·33)	August (0·56)	Mar. (134) Feb. (135)	Nov. (75)
Influenza	England and Wales [39], 1921-25	— d.	February (184)	August (14)	Dec. (2·75)	May (0·50)	—	—
Pneumonia	Liverpool, 1920-27	16,083 c.	December (164)	August (41)	Oct. (1·54)	June (0·33)	Mar. (133)	Dec. (77)
Meningococcal meningitis	England and Wales [39], 1921-25	— c.	April (129)	November (63)	Dec. (1·63)	August (0·76)	—	—
III.—Alimentary Infections.								
Enteric fever	Liverpool, 1889-1926	18,807 c.	October (151)	February (65)	{ Sept. (1·31) April (1·23)	December (0·69)	Dec. (115)	May (81)
,,	England and Wales [39], 1921-25	— c.	September (151)	March (66)	{ April (1·27) July (1·25)	November (0·72)	April (123)	Oct. (82)
,,	British Guiana, 1916-26	1,977 c.	March (157)	October (48)	January (1·88)	November (0·67)	—	—
Diarrhœa	Liverpool, 1889-1926	—	October (151)	February (65)	—	—	—	—
,,	1848-57	7,473 d.	August (258)	April (25)	July (2·70)	November (0·33)	—	—
Dysentery	,,	1,169 d.	August (247)	May (38)	June (2·11)	October (0·42)	—	—
,,	Russia [44], 1921	192,189 c.	July (350)	?	July (3·27)	?	—	—
,,	Singapore [45], 1923-25	2,041 d.	June-July (113)	October (65)	December (1·60)	October (0·69)	—	—
Undulant fever	Malta, 1906-26 [56]	11,844 c.	July (198)	February (42)	May (1·62)	August (0·73)	Feb. (125)	April (57)
Food poisoning	Europe [46]	Outbreaks	August (123)	January { 23	—	—	—	—
IV.—Autumnal Diseases.								
Diphtheria	Liverpool, 1881-1927	37,664 c.	November (122)	August (76)	Sept. (1·30)	April (0·84)	Mar. { 119) Ap. {	Sept. (68)

Chickenpox	Manhattan [42], 1919-23	8,776 c.	May (142)	September (51)	January (12·9)	August (0·71)	Feb. (158)	Oct. (50)
Scarlet fever	Liverpool, 1881-1919 ...	104,740 c.	October (140)	July (78)	August (1·39)	January (0·86)	Feb. (118)	Sept. (80)
,, ...	Manhattan [42], 1919-23	3,940 c.	May (164)	October (40)	December (1·76)	August (0·44)	Jan. (137)	July } (49) Sept. }
,, ...	London [47], 1892-97 ...	150,686 c.	October (152)	February (48)	May (1·28)	November (0·69)	Jan. (130)	Oct (74)
Erysipelas	Liverpool, 1891-1926 ...	30,141 c.	September (117)	July (84)	October (1·21)	January (0·83)	Feb. (123)	July (76)
Cellulitis	England and Wales [39], 1921-25	— d.	December (132)	August (73)	—	—	—	—
Rheumatic fever	London Hospital [48], 1873-81	738 c.	November (166)	April (57)	May (1·49) October (1·60)	August (0·74)	—	—
,, ...	Liverpool, 1877-1926 ...	1,944 d.	December (129)	September (81)	Nov. (1·26)	July (0·80)	—	—
Rheumatism in scarlet fever	Two Liverpool hospitals, 1909-12	142 c. / 3,559	Dec. (120) / May (126)	August (64)	—	—	—	—
Puerperal fever	Liverpool, 1891-1926 ...	2,049 c	November (115)	August (78)	February (1·21)	January (0·76)	Feb. (113)	August (78)
Vincent's angina	Liverpool, 1923-27) ...	76 c. (Bacterial)	Oct. (254) / Jan. (190)	May (32)	—	—	—	—
,, ,, ...	,, ...	164 c. (+ 78 Clinical)	October (455)	Feb. / May (25)	—	—	—	—
Catarrhal jaundice	England [49, 50] (Still and Gibson)	171 c.	November (179)	July (7)	—	—	—	—
Acute yellow atrophy ...	Lebert and Cockayne [51]	193 c.	Nov. (137) / Feb. (149)	September (38)	—	—	—	—
Diphtheria carriers... ...	London C. C., 1921-22 ...	281* c. / 10,106	May (120) / Oct. (135)	April (61)	—	—	—	—
Scarlet fever (return cases)	Liverpool, 1920-24 ...	331** c. / 14,318	May (139) / Dec. (126)	October (48)	—	—	—	—
V.—Insect-borne Diseases.								
Typhus fever...	Dublin [52], 1876-1921 ...	2,811 c.	December (145)	July (70)	Nov. (1·42) / Mar. (1·13)	April (0·69)	—	—
,, ...	Russia [53], 1912-20 ...	5,410,284 c.	January (146)	September (23)	Jan. and Feb. (1·77)	September (0·53)	—	—
Relapsing fever	,, ,, ...	1,307,805 c.	February (170)	September (55)	Jan. and Mar. (1·40)	August (0·77)	—	—
Malaria	Rome (Celli)	92,992 c.	August (228)	June (33)	July (3·45)	December (0·69)	—	—
,, ...	Athens [54], 1882-86 ...	14,027 c.	Aug. and Sept. (191)	May (28)	July (1·61)	December (0·63)	—	—
VI.—Avitaminoses, etc.								
Rickets (new cases)... ...	London [55]	348 c.	March (148)	August (48)	—	—	—	—
Tetany (infantile)	Germany [23]	240 c.	March (295)	July (0)	—	—	—	—
,, (adult)	,, [24]	576 c.	March (285)	Aug. and Sept. (19)	—	—	—	—
Serum rashes	Glasgow [25], 1913-14 ...	—	March (134)	August (9)	—	—	—	—
Scurvy...	Russia [44], 1921 ...	110,251 c.	June (191)	?	—	—	—	—
,, ...	Russia, 1923-25	132,980 c.	May (147)	December (44)	—	—	—	—
VII.—Venereal Diseases.								
Gonorrhœa	Sweden [45], 1923-25 ...	32,462 c.	August (172)	Mar. (83)	July (1·18)	February (0·81)	—	—
Primary syphilis	,, ,, ...	1,462 c.	January (137)	July (63)	—	—	—	—

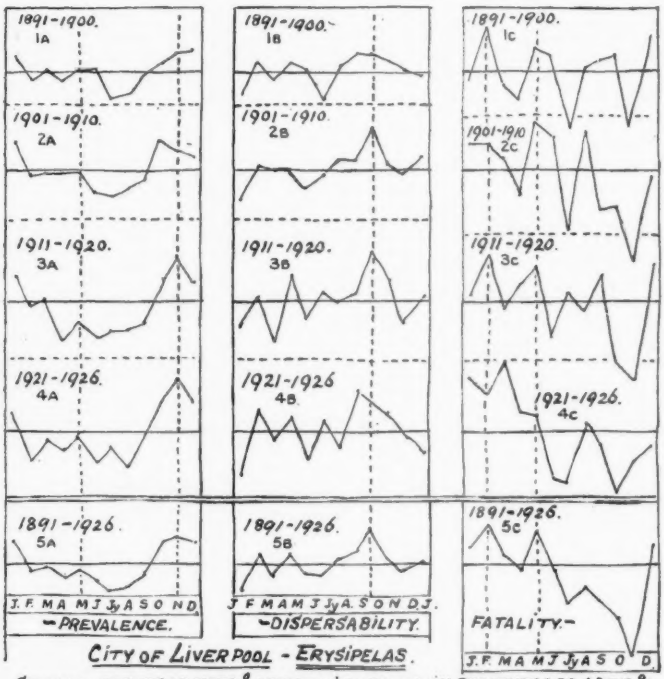
* 281 virulent carriers out of 10,106 children in L.C.C. Schools examined by Dr. J. G. Forbes.

** 331 cases giving rise to return cases out of 14,318 cases discharged from hospital.

† The months given under this heading are the first of the pair on which the ratio is based. Thus, December stands for December-January, etc.

epidemic in the central areas mainly to fly-borne infection, whereas case-to-case infection seems to play a much larger part in the outer areas; and this prosodemic infection is the sole agent in the outbreaks which occur in institutions where the prevalence is later than in the city at large.

Scarlet fever, formerly recurring in violent epidemics at regular intervals of three or four years (see Diagram XIII), was then always an autumnal disease: since then the peak months have shown greater scatter, and the modal month has moved on from September to October in Liverpool (see Diagram XIV). In fact, loss of



SEASONAL CURVES OF CASES & OF FATALITY RATES IN PERCENTAGES ABOVE & BELOW THE ANNUAL MEAN & OF INDICES OF DISPERSABILITY FOR DECENNIAL PERIODS AND FOR 1891-1926.

DIAGRAM XV.

regular periodicity, diminishing range of fluctuation of both periodic and annual cycles, i.e., flattening of the epidemic waves, retardation of the peak week, or month, and greater scatter of the modes of peak periods, are evidences of what I have termed "degradation of disease"—that is, its lapse from the epidemic phase. On the other hand, anticipation of the annual curve, associated with increased range of fluctuation, this latter shown by the curves of erysipelas in Liverpool (see Diagram XV), and both shown by diarrhœa in Liverpool during 1886 to 1905, are evidences of an opposite tendency which one may term "aggradation" of a disease.

In short then, changes in seasonal prevalence may be evidence of an alteration in the modes of conveyance, or of some secular change in the relationship of virus and host, which I have termed aggradation and degradation of disease.

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Discussion.—Sir LEONARD ROGERS (remarking on diagrams and maps which he showed to illustrate the seasonal incidence of disease in relation to climatic conditions in India), said that pneumonia showed by far the highest incidence in the Sind and North-West Frontier Provinces, and became steadily less from there to the south of Madras and Burma in relationship to diminished diurnal variations in temperature, and increase of the winter minimum temperature and absolute humidity. In the high incidence areas there was a diurnal variation of the cold weather temperature of over 30° F., accompanied by a minimum temperature of about 30° F., and an absolute humidity of ·250 to ·300, all tending to produce chills predisposing to pneumonia.

Small-pox in India fell to a low minimum with the maximum rise of absolute humidity in the monsoon months from June to September, and rose steadily during the dry cold and hot weather seasons of low absolute humidity, except in Madras which was not materially influenced by the south-west monsoon. In this country small-pox increased during the winter months of low absolute humidity and low temperature, which here ran a parallel course, but Indian experience showed that the absolute humidity was the important factor.

Cholera fell to a minimum in the dry cold weather, except in Assam, Bengal and Madras, where it was very high in the cold season, and his maps showed that this was due to the absolute humidity never falling below ·400 in these areas, as it did in the rest of India, so that cholera was never widely epidemic with an absolute humidity below ·400, and it rose in March to May in northern India with the increase of the absolute humidity over that point, being thus a recrudescence and not a spread from Bengal, as held for a century past.

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Plague fell to its seasonal minimum in the hot weather with a mean temperature of 85° F. and over, and with low saturation deficiency, as first shown by St. John Brooks, and in the Deccan and Central Provinces it rose during the monsoon months, as the mean temperature then fell below 80° F., but in Northern India, where the mean temperature remained at a higher level during the monsoon months, plague continued at a low level, and only rose about December with the great decline of the mean temperature.

Thus in all these diseases the seasonal incidence could be explained on climatic grounds, and the absolute humidity, essentially a measure of combined heat and moisture, proved to be the most important factor in most of them.

Sir WILLIAM HAMER said that the rise of enterica in May had sometimes in the past been found to be due to cockles. Bulstrode, in his Shell-fish Report of 1911 (p. 104), had discussed occurrences in London, of cockle outbreaks following upon the Easter and Whitsuntide Bank Holidays. There was a clearly demonstrated instance of this sort in 1902. With regard to the Registrar-General's figures for 1921-25, it was open to question as to how far these could be held to relate to typhoid fever. Before the war the percentage of error in diagnosis was 25-30 per cent., and since 1911 the autumnal rise in prevalence had practically disappeared, so that the percentage of cases diagnosed in error was presumably quite considerable at the present time.

Dr. PERCY STOCKS said that the "index of dispersability," as defined by Dr. Stallybrass, was really the same thing as the ratio between successive ordinates of the epidemic curve which Dr. Farr had measured. If the assumption was that the cases in the second period arose from those in the first, it would be more logical to calculate the index from periods whose centres were separated by the mean or modal value of the incubation period of the disease rather than by an arbitrary period of a week. Otherwise the indices for diseases having different incubation periods were not really comparable. Caution was also necessary in drawing conclusions from the curve of such an index, because it was partly a function of the number of susceptible persons left in the population, which was changing to an unknown degree as the epidemic progressed.

In connexion with the correlation between influenza and air temperature, Miss Hilda Woods had shown in a recent paper that, whereas the weekly pneumonia mortality in the last quarters of the years 1850-89, when influenza was not prevalent at that season, gave an inverse correlation of 0·46 with mean air temperature in the preceding week, this was increased to 0·71 in 1903-14 when influenza was prevalent. A few years ago he (Dr. Stocks) had shown that weekly mortality from circulatory diseases in London in October-November during 1900-14 and 1919-25 gave a correlation of — 0·44 with mean air temperature in the same week, and after correcting for air temperature by partial correlation there remained a small correlation of 0·19 with the barometric height in the same week.

Dr. C. O. STALLYBRASS (in reply) said that Sir Leonard Rogers was fortunate in having found in absolute humidity a measure which represented the combined effects of temperature and relative humidity and so to have escaped the necessity of engaging in the calculus of partial correlation. He could not agree with Sir William Hamer that, so far as Liverpool was concerned, the spring rise of enterica was due to the consumption of shellfish: personal inquiries there over a number of years into every notified case revealed no rise in May of cases due to shellfish.

He agreed with Dr. Stocks that the description of the index of dispersability as representing the number of cases to which one case gives rise was rather crude. It might be more precisely defined as the rate of increase or decrease when comparison is made between two equal periods. It was not practicable to base it on the incubation period, as in some diseases that was indefinite or unknown. Indices of dispersability based on one unit, e.g., a week, must not be compared with indices based on another unit, e.g., a month.

In calculating fatality-rates, it was necessary to take into account the mean period between notification and death. In measles, scarlet fever and diphtheria, this was short and might be neglected: in whooping-cough, however, and probably in enteric fever and undulant fever, it was longer and the lag-period must be offset by a set-back of the deaths. In the absence of direct evidence on this point in a given disease, the set-back which gave the smoothest curve was probably the best.

Section of Medicine.

President—A. F. Hurst, M.D.

[*March* 27, 1928.]

Note on Idiosyncrasies and Abnormalities in Human Beings.

By F. Parkes Weber, M.D.

Abstract.—Idiosyncrasies are the expression of abnormal mental or physical reaction towards "agents." An attempt is made to indicate the position of idiosyncrasies in a classified scheme of all abnormalities; but bodily idiosyncrasies due to an "allergic" or *hypersensitive response to agents* are mainly considered in the present paper. Migraine and mucous colic ("colitis mucosa") are discussed in connexion with Freeman's view of food idiosyncrasies, hay fever, asthma, urticaria, angioneurotic œdema, &c., as manifestations of an "immunological abnormality or defect."

The hypothesis that idiosyncratic hypersensitiveness towards physical agents, such as light, heat, cold and mechanical trauma, may in reality be the expression of reaction towards a histamine-like body, or protein of some kind (virtually a "foreign protein") liberated in the tissues by the physical agent in question, is referred to, according to the works of W. W. Duke and Sir Thomas Lewis and his co-workers, and the writings of Sir Humphry Rolleston on the subject. A somewhat analogous explanation is suggested for the following conditions: an abnormally hypersensitive (eczema-like) reaction towards formalin lotions; epidermolysis bullosa; constitutional factitious urticaria in otherwise healthy individuals not suffering from ordinary urticaria; excessive liability to chilblains; so-called "erythrocyanosis" of the lower parts of the legs in girls and young women, and some cases of Raynaud's disease.

The significance of eosinophilia in cases of dermatitis herpetiformis and pemphigus is also alluded to.

All idiosyncrasies, if sufficiently marked to deserve the name, are abnormalities, but not all abnormalities are idiosyncrasies. Abnormalities, congenital or acquired, may be divided into the following classes:—

(1) Abnormalities in structure, that can be demonstrated by macroscopic or microscopic examination. This class can, of course, be subdivided almost to any length.

(2) Abnormalities that cannot be demonstrated by macroscopic or microscopic examination, but are manifested by abnormality in action, or in functional reaction towards an "agent."[1] This second class may be subdivided into: (*a*) Those in which the mind and central nervous system are concerned, which include congenital deficiencies in the special senses, cases of defect and peculiarity in the perception of mental and moral pains and pleasures, and intellectual and moral defects and peculiarities of various kinds and degrees;[2] (*b*) functional abnormalities in the vegetative nervous system (including exaggerated examples of so-called "vagotonia") or in the endocrine system or in both; (*c*) metabolic abnormalities, including Sir Archibald Garrod's "inborn errors of metabolism" (congenital hæmatoporphyrinuria, alcaptonuria, cystinuria, etc.); and lastly, (*d*) abnormal reactions of body or mind towards "agents" in otherwise normal individuals. The examples of this last subdivision may be termed *idiosyncrasies*, and it is practically in regard to these that my remarks at present will be confined.

1 In this paper I use the term "agent" to signify something that excites a "reaction," as I used it in 1903, in a "Note on Action and Reaction in Pathology and Therapeutics," *St. Bart. Hosp. Reports*, London, 1903, xxxix, p. 139.

2 It is possible that cases described as "congenital neurasthenia," and "(congenital) psychasthenia," and some cases of "effort syndrome" really belong to (*a*) or to (*b*) or to both these two subdivisions.

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It is true that the subdivisions of the above two classes may intermingle, and also that examples of combined congenital structural and congenital functional abnormalities are common, but for my purpose the classification is useful, though of course it must be remembered that congenital abnormalities in function are doubtless often in reality structural as well, though we cannot yet recognize the structural abnormalities in question.

IDIOSYNCRASIES.

In regard to *mental idiosyncrasies* it is almost certain that some have resulted from mental "complexes" and associations with subconscious recollections of disagreeable incidents in the past, but Sir Humphry Rolleston mentions [1] two patients, of whom Dr. Mackenzie Wallis told him, both of whom had an antipathy to cats, and in both of them skin tests showed abnormal sensitiveness to cat extract, and minute injections of that extract brought about de-sensitization, so that the antipathy disappeared. Abhorrence of cats is said to have been noted in, amongst others, Napoleon, Wellington, and Lord Roberts,[2] and from a modern psychological point of view it would be interesting to know whether women show a horror of cats as often as men do. One would expect them more often to show a horror of big dogs, which are the special favourites of men. But this is quite uncertain.

Bodily idiosyncrasies, as distinguished from mental idiosyncrasies, are manifested by the occurrence of abnormal bodily reactions towards "agents," especially chemical agents, such as drugs,[3] foods, and minute quantities of "foreign proteins," in otherwise normal individuals. The abnormality in reaction may be due to excessive sensitiveness ("allergic" or "hypersensitive" response), or more rarely to abnormally slight sensitiveness, that is to say, excessive tolerance.[4] Abnormal tolerance may in many instances be acquired, as it is by all smokers towards tobacco (at least as regards ordinary smoking of tobacco), and by some towards alcoholic drinks, and, as it has been said, towards arsenic, by modern Styrian peasants. The acquisition of such (more or less marked) tolerance towards poisons is sometimes termed "Mithridatism," after Mithridates the Great, King of Pontus (120-63 B.C.), who is said to have investigated it.[5] But I shall refer here only to idiosyncrasies manifested by hypersensitive or allergic response, which are better known and probably more important than those manifested by abnormal tolerance. The hypersensitive response may take the form of urticaria, cutaneous erythema, asthma, vomiting, diarrhœa, etc.

Of idiosyncrasies due to *hypersensitiveness*, or *allergy*, in reaction, the most interesting are perhaps the examples of congenital origin. The characteristic of the heredity observed in regard to these idiosyncrasies is that in different members of the same family the manifestations of the familial hypersensitiveness may be quite

1 Sir Humphry Rolleston, *Idiosyncrasies*, London, 1927, p. 24.

2 Mentioned by various writers, including Gould and Pyle. *Cf.* F. Parkes Weber, *Medical Press and Circular*, London, 1920, clx, pp. 478-479. Unnatural fear of cats is referred to by Aristotle (*Ethics*, Book vii, Chapter 5), and fainting at the sight of cats is said to be mentioned by a French medical writer, Quercitanus (Joseph Duchesne), who lived about 1544-1609—I suppose in the section "on fear" in his *Diaeteticon Polyhistoricon*.

3 Naturally, what is spoken of as the normal action of a drug is, in most cases, in great part or entirely, what I have termed the normal reaction towards the drug, as I explained in regard to digitalis in my article in *St. Bartholomew's Hospital Reports*, 1903, *loc. cit.*

4 It is likewise possible that the abnormality in response may in some cases be a qualitative one rather than a quantitative one.

5 See F. Parkes Weber, "Active and Conservative Vital Reaction, especially in regard to Idiosyncrasies," *Clinical Journal*, London, 1909, xxxiv, p. 175. A superficial analogy may be drawn between physical tolerance towards foods and drugs, and mental tolerance towards exciting "agents." There are apathetic individuals who react no more than is absolutely necessary, and hypersensitive persons who are "touchy." Acquired mental hypersensitiveness is illustrated by persons with mental "complexes."

different; in one member there may be hay-fever, in another asthma, in another tendency to migraine, and in another to a kind of eczema. The manifestations in a single individual may be different at different periods of life, or one manifestation may alternate with another during the same period. In a paper on the "Pathological Mechanism of the Asthma Syndrome," Dr. John Freeman [1] took as his subject the family of diseases of which asthma is such a conspicuous example. He says that the life history of an asthmatic patient frequently starts with food difficulties. There may, for instance, be difficulties in weaning. Special food idiosyncrasies may be accentuated to almost any extent and may persist through life. He says, however, that these often fade away, and in early school days there may be eczema, and then, perhaps, through early adult life, hay-fever, and in old age, bronchial asthma. An attack of eczema may, he remarks, alternate with headache, and asthma with angioneurotic œdema or urticaria.

In common, with various others, I think that angioneurotic œdema is similar in nature to urticaria, but I believe that in the former the process starts in the looser subcutaneous tissue, so as to give rise to a large subcutaneous swelling, whereas in urticaria the same process, starting in the cutis, can produce only an ordinary urticarial weal; the term "giant-urticaria" should be reserved for those cases in which an ordinary urticarial weal in the cutis spreads to the subcutaneous tissue, so as to produce the more extensive swelling of angioneurotic œdema.

The Prausnitz experiment, referred to by Freeman, proves that these cases are really due to hypersensitiveness. Thus, blood-serum from a hay-fever patient may be injected into a normal man's skin, so as to sensitize the area injected towards pollen (which produces hay-fever). Then a local reaction in this sensitized area can be produced either by injecting pollen into the area itself, or by injecting pollen into the blood-stream, which carries it to the area in question and sets up the local reaction. A dose of pollen which, injected into the blood of a hay-fever patient is just sufficient to upset him, would probably not affect a normal man, even if a hundredfold increased. A normal man, says Dr. Freeman, after a week-end holiday in the country during the hay season will have sufficient pollen in his circulating blood to cause an urticarial reaction when a drop is injected into the skin of a hay-fever patient.

I will not attempt to discuss the various idiosyncrasies towards "foreign proteins," as manifested by hay-fever, asthma, urticaria, angioneurotic œdema, attacks of eczema, diarrhœa, vomiting, migraine, etc. According to Dr. Freeman (loc. cit.) the inherited factor that connects them is an *immunological abnormality or defect*. It is, therefore, analogous to the defect which makes some persons more liable to tuberculosis (and to a special local incidence of tuberculosis) than other persons.

The analogy of migraine with known allergic reactions has been much discussed. Recently J. S. Diamond [2] examined thirty-five cases with cephalic and abdominal migraine symptoms, and the results of his examination indicated the presence of a functional abnormality of the liver ("dysfunction" of the liver, as he terms it). In 91 per cent. of these cases there was a "latent icterus," with a retention of blood-bilirubin, equivalent to 1 to 5 Hijmans van den Bergh "units." The urobilinogen was also increased. As an explanation of these cases, which he classes under the heading "hepatic migraine," he suggests the inability of the liver to detoxicate putrefactive substances, that is say, putrefactive substances derived from animal proteins in the intestinal canal, and he attempts to explain the resulting symptoms as an allergic phenomenon, that is to say, an allergic reaction. He speaks also of

[1] J. Freeman, *Lancet*, 1928 (i), pp. 288-290.
[2] J. S. Diamond, "Liver Dysfunction in Migraine," *Amer. Journ. Med. Sci.*, 1927, clxxiv, pp. 695-702.

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beneficial therapeutic results obtained by abstinence from animal proteins, "changing the intestinal flora," and relief from spastic constipation.

In regard to food idiosyncrasies it should be remembered that enormous advances in recent times have been made in knowledge regarding allergy and hypersensitiveness towards "foreign proteins." Minute quantities of foreign fats and proteins are more than was formerly supposed taken up in an unaltered state from the alimentary canal into the blood-stream and tissues of the body (compare the results of tests by Abderhalden-like methods), and doubtless that is so in some individuals more than in others. Certainly also the degree of reaction to the same amount of foreign protein varies greatly in different individuals. It is quite natural, therefore, that occasionally —"Quod cibus est aliis, aliis est atre venenum" (I quote this in spite of the fact that in classical Latin the *atre* should be *atrum*, and then the hexameter verse would be spoiled)—or, "What's one man's poison is another's meat or drink." (Beaumont and Fletcher's *Love's Cure*, Act III, Scene 2.)

The so-called "colitis mucosa," or rather, "mucous colic," which especially affects women of nervous temperament, between the ages of 20 and 40 years, and which was formerly regarded as a secretory neurosis, just as asthma was supposed by some to be of only nervous origin, seems now to be still more analogous to asthma, inasmuch as it appears to be, like asthma, of allergic origin, due to hypersensitiveness towards certain foods. Treatment by the exclusion of the foods in question has led to excellent results in E. Hollander's hands [1].

Before leaving the subject of food idiosyncrasies I would suggest that writers on proposed "food reforms" may occasionally have been biased in their views and proposals by something like food idiosyncrasies in their own persons.

In regard to idiosyncratic *hypersensitiveness towards physical "agents,"* such as light, heat, cold and mechanical trauma, Sir Humphry Rolleston [2] quotes W. W. Duke's hypothesis that the patient may be sensitive to a protein body (virtually a "foreign protein") liberated from the cutaneous cells by the action of some physical factor, whether heat, cold, sunlight or mechanical trauma. Rolleston likewise quotes Sir Thomas Lewis and Grant's observations (supporting Duke's hypothesis) that mechanical and other forms of irritation normally liberate a histamine-like body from the cells of the skin, with resulting production of urticaria. Lewis (1927) concluded that the skin reactions produced by physical agents precisely resemble the anaphylactic skin reaction and that produced by injection of histamine—also that the liberation of the histamine-like substance is a fundamental factor in the anaphylactic reaction.

It has occurred to me that a special cutaneous hypersensitiveness, shown by an eczema-like reaction to formalin lotions in some persons, may really be due to local hypersensitiveness to protein substances produced by the action of formalin on the skin, rather than directly towards the formalin itself.

It seems to me also that the rare familial disease known as "epidermolysis bullosa" (formerly called "traumatic pemphigus," or "familial traumatic pemphigus") may be partly explained in the same way. In this disease the bullæ are excited by local slight mechanical trauma. One might suggest that the local allergic response to the slight trauma, which is manifested by the formation of a bulla, is due really to some chemical change set up by the trauma and resulting in the local formation of a (virtually) foreign protein. (Analogously the factitious urticaria so easily produced in most children with urticaria pigmentosa, and also in some apparently healthy individuals, might be regarded as the manifestation of an allergic local response to some substance set free by very slight mechanical traumata.)

1 E. Hollander, *Amer. Journ. Med. Sci.*, 1927, clxxiv, p. 495.
2 Rolleston, *op. cit.*, pp. 50-53.

R. Pollard, in a paper on epidermolysis bullosa hereditaria in 1916[1] favoured the view that the lesions were due to local hypersecretion of serum, and that the disease was related to urticaria and angioneurotic œdema. P. Linser,[2] in 1907, had suggested that the disease was a primary vasomotor neurosis, leading to necrosis, of which the disintegration products were locally irritating.

The general conclusion arrived at from the writings on *eosinophilia*—including both local (or tissue) eosinophilia and general (or blood) eosinophilia—by E. Schwarz, Schlecht and Schwenker, Ahl and Schittenhelm, and Homma, is that it constitutes part of a general biological protective reaction of the body against foreign proteins and abnormal disintegration products of the body's own albumin[3]. Dermatitis herpetiformis and pemphigus are often accompanied by a high blood-eosinophilia, and this suggests at first sight that the vesicular and bullous lesions of these diseases might be allergic manifestations of reaction towards some "foreign protein." The high blood-eosinophilia in such cases may, however, only be due to absorption (from the lesions) of disintegration products, which, in regard to eosinophilia, might exert an effect similar to that of foreign proteins.

The above mentioned investigations of Sir Thomas Lewis and his co-workers regarding the local effects of traumata and physical "agents" on the skin throw a flood of light on various puzzling problems. They have shown that as the result of certain local physical stimuli or agents a histamine-like body may be set free in the tissues, giving rise to urticarial or other local reactions. Something of this kind perhaps takes place as the result of local exposure to cold in persons liable to chilblains, and it should be remembered that a chilblain-like reaction towards cold is so easily produced in some individuals as to constitute a real idiosyncrasy; the pinnæ of the ears in such individuals are often especially involved.

There is no hard and fast line between chilblainy conditions, due to a truly idiosyncratic predisposition and frost-bites in normal individuals. Cases might be arranged in a series, at one end being those which are the expression of the reaction towards the almost overwhelming action of the physical agent (cold), at the other end being those in which as an idiosyncracy there is a definitely abnormal reaction to relatively slight exposure to cold. In the latter cases one may well suggest that the local action of cold sets a chemical substance free in the tissues which brings about the turgid swelling and itching characteristic of chilblains, to some extent analogous to the local phenomena of urticaria and "serum-rashes."

I would suggest that the erythematous or chilblainy condition of the legs in girls and young women which I have elsewhere discussed might similarly be explained as representing an abnormal reaction—that is to say, a reaction occurring only in abnormally predisposed or idiosyncratic subjects—towards the local application of cold, the local effects being favoured by the scanty clothing of the affected parts, in accordance with prevalent fashion.[4] The condition in question is now being described on the Continent under the heading, *erythrocyanosis crurum puellaris frigida*, but A. V. Karwowski[5] suggests that it should be termed "erythrocyanosis

1 R. Pollard, *Dermat. Zeitschr.*, Berlin, 1916, xxiii, p. 385.

2 P. Linser, *Arch. f. Derm. u. Syph.*, Wien, 1907, lxxxiv, p. 369.

3 F. Parkes Weber, "Eosinophilia," *Med. Press and Circular*, London, 1927, clxxv, pp. 152-154.

4 F. Parkes Weber, "Note on a kind of Chronic Erythema of the Legs in Girls and Young Women," *Brit. Journ. Derm. and Syph.*, London, 1925, xxxvii, pp. 259-262; *idem*, "Two Diseases due to Fashion in Clothing," *Brit. Med. Journ.*, London, 1925 (i), pp. 260-262; *idem*, "Two Cases of Chilblainy Condition of the Legs," *Proc. Roy. Soc. Med.*, 1927, xx (Sect. Derm.), pp. 61-65 and *Brit. Journ. Derm. and Syph.*, London, 1927, xxxix, pp. 259-262.

5 A. V. Karwowski, *Dermat. Wochenschr.*, Leipzig, 1927, lxxxv, p. 1161. See also A. Alexander, "Erythrocyanosis crurum puellaris," *ibid.*, 1927, lxxxv, p. 601. The term *erythrocyanosis cutis symmetrica* has also been employed.

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crurum feminarum (frigida)," as it has been known to recur in middle age. Karwowski holds that cold to the legs and internal generative organs due to modern clothing plays an important part in the causation. I would, by the way, here note that I am sure those authors are wrong who assert that the condition never proceeds to definite thickening and hardening of the affected skin. The condition may be associated with, but is to be distinguished from, a tendency to ordinary chilblains. It may be associated with, but is also definitely to be distinguished from "acrocyanosis" or so-called "bad circulation in the extremities" of young persons, which occurs in males as well as females. If the above-mentioned modern name be adopted, the epithet *frigida* should not be omitted, for it distinguishes the condition from (tuberculous) erythema induratum ("Bazin's disease")—with which severe cases have doubtless often been confused—and from erythromelalgia. It is to some extent analogous to chlorosis (which has now become rare), in so far as it almost only affects youthful females and only those who have an abnormal predisposition to it and in whom the exciting cause (whatever it may be) is at work. The exciting cause in the erythrocyanosis cases is the local action of cold, and the predisposition consists in the *idiosyncrasy* to respond to local exposure to moderate cold in the manner in question and not necessarily by the development of ordinary chilblains.

It is also possible that some, though not all, cases of so-called Raynaud's disease and symmetrical gangrene of extremities (especially the tips of the fingers and toes) may be explained in a similar manner, as representing an idiosyncratic effect of cold in certain individuals—the cold setting free a chemical substance in the affected parts, which leads to local angiospasm and further in some instances to local thrombotic vascular occlusion, with more or less gangrene as a result. Theoretically it is possible for the unfortunate subject of such an idiosyncrasy to live in the tropics without ever suffering from the effect of his idiosyncrasy, until he leaves for a colder country, when he may suddenly present signs of spontaneous symmetrical dry gangrene in the fingers and toes from a first attack of so-called Raynaud's disease. Possibly C. C. Choyce's remarkable case [1] of "Symmetrical Gangrene of Both Legs, Fingers of Both Hands, and Nose" was an example in question, but the occurrence of "Raynaud's disease" is not altogether unknown in hot countries.

My remarks in this paper should be considered merely as a collection of marginal jottings to various writings on the subject.

Mr. FRANK COKE said that amongst the large number of asthmatic patients seen by him, some were extraordinarily sensitive to otherwise harmless proteins.

Several showed an idiosyncrasy to brazil nuts in an extreme degree. One such patient sat down to dinner believing that there were no brazil nuts in the house. At the end of the meal he became seriously ill, with diarrhœa and vomiting, and an exhaustive inquiry revealed the fact that there was some brazil nut in a mince pie which he had taken.

Another patient became comatose after eating an apple; smaller quantities would paralyse her accommodation for some hours, and merely walking through an orchard was sufficient to bring out an urticarial rash.

He had another patient, managing director of a large business, who was becoming so strange and irritable in his ways that some mental disturbance was feared. On being tested to a long series of proteins he gave a large reaction to potato, and after giving up this vegetable entirely he quickly returned to normality.

He (Mr. Coke) believed that these idiosyncrasies were due not to anything lacking in the body or to a want of protection, but to an additional specific "adzyme," as he had called it, in the blood. It was impossible to imagine that individuals were sent into the world with their outfit complete, except that they had no protection against such out-of-the-way proteins as

[1] C. C. Choyce, *Proc. Roy. Soc. Med.*, 1911, iv (Clin. Sect.), p. 77. There was apparently hæmoglobinuria in this case at the commencement of the attack.

caviare, brazil nuts or apples. What happened was that the body had a surfeit of some particular protein and dealt with it, but in the process the adzyme became specific for that protein. After an incubation period, if more of the same protein was taken, an immediate aggregation or flocculation of the colloidal particles of the blood took place, causing asthma and its many fellow complaints.

The glut of a protein and then no more for a time, allowing for an incubation period, explained the common sensitizations to crabs, lobsters and rarer foods, and, of course, pre-eminently, the common hay fever due to pollens.

New Zealand Views on Goitre.

By D. W. CARMALT JONES, M.D., F.R.C.P. (Dunedin, N.Z.).

ABSTRACT.—Simple goitre is highly prevalent in New Zealand, and there is considerable incidence of toxic goitre. The ætiology of simple goitre seems fairly well established, and an attempt is being made to apply the data from simple goitre to the problems of toxic goitre.

Endemic goitre is of great antiquity among the Maoris, and has been described among Europeans for about fifty years. It occurs in both men and animals. At five years its incidence is similar in boys and girls, later it decreases in boys but increases greatly in girls. It is often hereditary, and many children are born goitrous. In children it is generally small, but may enlarge and cause pressure, myxœdema and toxicity. Its incidence varies greatly in different districts.

The only cause found consistent with this variation in distribution is lack of iodine in the soil. An inverse ratio has been demonstrated between the iodine content of the soil and the incidence of goitre in school children in thirty-three districts. The iodine content of the soil is reflected in the food raised upon it.

The daily iodine intake was estimated at 35 microgrammes in a non-goitrous, and at 20 microgrammes in a goitrous district.

The amount of iodine involved is infinitesimal, and its intake can be ensured by the use of salt for ordinary consumption, which contains four parts per million of potassium iodide.

Toxic goitre is also frequent: in this connexion, the influence of iodine on the thyroid has been investigated. If starved of iodine the thyroid adapts itself either by increasing its colloid or by a diffuse hyperplasia, both may occur in different parts of the same gland. Simple goitre is the response of the healthy thyroid to iodine deficiency, the responding areas may be diffuse or adenomatous, and degenerations may occur. Such goitres may be treated with iodine, in children re-adjustment to the increased intake is readily made, but in adults long accustomed to a low intake, excess often causes too great hormone production, with toxic symptoms, hence the minimal dose alone is permissible in iodized salt.

Goitre stored with iodine at low pressures may become toxic under stress, and this may be precipitated by iodine. The prevalence of toxic goitre may be partly due to the prescription of iodides in therapeutic doses for common ailments.

Diffuse colloid goitre may subside under physiological iodine, the adenomatous is more prone to toxic symptoms and may go on to secondary Graves' disease or to myxœdema. Diffuse hyperplasia is a possible manifestation of iodine deficiency as primary Graves' disease. Lugol's solution probably allows of a temporary storage in this condition. Iodine has certainly some bearing on the problems of toxic goitre.

GOITRE is highly prevalent in New Zealand. As in all countries where that condition is endemic, it presents two series of problems, those of simple or non-toxic goitre, and those of toxic goitre. The two are discussed separately here. It is believed that the essential ætiology and pathology of simple goitre, as regards New Zealand, are fairly well understood, and that the condition can and will be prevented when the medical profession and the public are sufficiently alive to the facts. Toxic goitre, on the other hand, is one of the most difficult subjects in medicine; although of common occurrence in New Zealand, it is only recently that any coördinated study of it has been attempted there; at present an effort is

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being made to apply the knowledge derived from the study of simple goitre to the more complex problem, while it is fully realized that there are other important factors involved. The views put forward on toxic goitre are therefore admitted to be speculative.

Endemic Goitre.

Simple or Non-toxic Goitre.

Historical.—Goitre has been prevalent in New Zealand in some districts as far as Maori tradition goes back. In the Urewera district in the North Island, an inland and inaccessible forest region, where the most primitive existing Maoris are found, the condition is common, and was found in 18 per cent. of 300 persons examined in a recent survey [1]. It is there known as "tenga," and this word has been used for it for many generations. The Coastal Maoris, on the other hand, for reasons which will appear, are free from goitre.

How long it has been observed among Europeans is not clear, but it has been recorded among them for at least half of the short period of the British occupation. New Zealand was annexed to the Crown in 1840, and serious settlement began in the South Island in 1848. In 1880 Dr. Hacon was appointed to the Christchurch Mental Hospital and observed endemic goitre there, which occurred both in old residents and in recent arrivals. He reported that the attendants' children suffered from it, that a child was born goitrous, and that a death had occurred from Graves' disease [2].

Extent and Importance.—In view of the existence of goitre among the indigenous Maoris and its occurrence among Europeans within thirty years of their occupation, it is likely to be prevalent at the present time. It is not confined to human beings, but occurs among horses, cattle, sheep and dogs, among domesticated animals, and among trout kept in captivity. The investigations upon animals are not complete.

During the War 1,500 men were rejected for military service on account of goitre, out of about 130,000 men examined, and goitre is far commoner in women than in men [3].

The principal investigations on man are those initiated by Hercus and Eleanor Baker in Canterbury and Westland, and since extended to the whole country, on school children. These are taken as the most accessible and the most representative section of the population which is available. The goitres were classified in four grades, "incipient," "slight," "medium" and "large," as was done by Marine and Kimball in America [1].

In all, 92,713 children were examined [4], of whom 28,207 were found to have goitre, nearly all incipient or slight, a percentage of 30·4, nearly one child out of every three examined had a goitre. The age and sex-incidence were closely studied in 1,666 children at Timaru; great numbers arrive at school at the age of 5, with goitre, many of the parents, brothers and sisters of these children are goitrous. The incidence at this age is similar in both sexes, boys being slightly more affected than girls; with advancing age the boys' goitres often disappear, but the incidence among girls increases very greatly.

A strong hereditary influence is thus indicated. In the Christchurch Maternity Hospital 60 per cent. of the mothers are said to be goitrous, and 8 per cent. of the babies born have enlarged thyroids. It can, therefore, hardly be doubted that goitre, unless checked, will increase. It is evident that some factor in New Zealand life induces goitre in the young, and the question arises why one child is affected and two escape. It is partly to be explained by heredity. It will be shown later that the goitrous thyroid displays various histological changes in response to a particular stress. Similar changes are found in thyroids which are not

actually enlarged. It appears probable that all thyroids exposed to the same stress undergo these changes, but that persons of goitrous heredity show greater enlargement, so that goitre in the second generation is likely to be more manifest than in persons whose parents were not affected.

In the great majority of cases this enlargement to meet a particular stress is moderate, and if it meets it satisfactorily there is no more than a cosmetic disability, but that is not always the case. Some teachers state that the mental acuity of school-children is lower in districts where goitre is prevalent than in others [1]. Also, goitrous patients are open to certain special risks: the thyroid may enlarge sufficiently to cause pressure symptoms; hæmorrhage, fibrosis and atrophy are all liable to occur in it, after which any degree of hypothyroidism up to actual myxœdema may be found [5]. Cretinism is at present rare, but exists; judging from Swiss experience, the risk of it is serious. Finally, a considerable number of simple goitres become toxic.

Endemic goitre in New Zealand thus presents a very large and very important problem.

Ætiology.—Hacon [2], the first to report the condition, regarded it as "miasmatic," and advised concrete foundations in houses and the lowering of the subsoil water. Colquhoun (1910) [6], discussed living matter, lime, inorganic substances and glacier-débris as possible causes and dismissed them all. He favoured a "radioactive" substance, probably present in drinking water. Drennan (1919) [7] noted the similarity of conditions in New Zealand to those reported by Marine in America; the latter had cured goitrous trout with iodine, and had found fœtal pigs born goitrous when the mother's diet was deficient in iodine, which was corrected by appropriate feeding of the sows, and he had also found a large number of schoolgirls with goitre in the Great Lakes District, and had prevented or arrested the condition with iodine. Drennan found goitrous trout in New Zealand, lambs born in a state similar to that of Marine's pigs, and a high incidence of goitre in the population. He advised the assay of food stuffs for iodine. This is the first recorded suggestion of iodine deficiency as a possible cause of the New Zealand goitre. Hercus (1921) [1, 3, 9], working with Eleanor Baker on goitre in school-children in Canterbury and Westland in the survey mentioned above, found that the incidence in Christchurch was very high, but that in the neighbouring district of Lyttelton it was lower by 20 per cent. It was found that there was a slight contamination of the Lyttelton artesian wells from the sea, and that the Lyttelton water contained more chlorine and iodine than that in Christchurch. Hercus independently suggested that this comparative immunity was due to the iodine in the water derived from a marine source. Since that time Drennan and Hercus have been colleagues and in close co-operation, and the present views on goitre in New Zealand are entirely due to their work.

The ætiology has been closely studied by Hercus. The striking fact about New Zealand goitre is that its incidence is exceedingly variable in different parts of the country. This was noted both by Hacon [2] (1892), and Colquhoun [6]; the latter published a spot map of endemic areas in 1910. Any theory as to the ætiology must account for this fact.

There are four "centres" in New Zealand with which groups of "land districts" are associated; thus in the North Island, Auckland is the centre for Auckland Province in the N.W., Wellington is the centre for Wellington, Taranaki and Hawke's Bay Districts in the S.E. In the South Island, Christchurch is the centre for Canterbury, Nelson, Marlborough and Westland in the N.E., and Dunedin for Otago and Southland in the S.W.

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The results of the examination of school-children in the four districts are as follows:—

Centre	No. of examinations	Cases of goitre	Percentage
Dunedin district ...	17,886	4,763	26·6
Christchurch district ...	18,133	10,260	56·3
Wellington district ...	38,099	10,253	28·0
Auckland district ...	18,595	2,931	16·0

If smaller districts are taken, the differences are still more striking, as is shown below, but the bad eminence of the Christchurch centre is immediately evident, and is borne out in many directions; thus of the 1,500 recruiting rejections 1,000 were in the Canterbury Military District. Again, in the Christchurch Mental Hospital, whose inmates of course often live for very long periods on Canterbury soil, Hercus found among some 800 patients nearly 60 per cent. of the women and 50 per cent. of the men with goitre, about 20 per cent. showing huge deformities. In contrast to this Beattie [6], in 1910, reported that in sixteen years he had only seen three cases of goitre at the Auckland Mental Hospital.

There are six theories current to account for the occurrence of endemic goitre in general, namely: (1) Fæcal contamination of water; (2) radio-activity of water; (3) salts dissolved in drinking water; (4) general diet; (5) geological formation; (6) iodine deficiency in diet. Hercus [1, 8, 9] has examined these exhaustively to determine how far they are applicable to New Zealand. His more intensive work has been done in Canterbury. His conclusions are set out below. It is to be remembered that his criterion of prevalence is the incidence among school children.

(1) *Fæcal Contamination of Water.*—The incidence of goitre in Christchurch school-children is very high (over 60 per cent.) but the city is well-sewered; the water-supply is artesian and of a high standard of bacteriological purity. In Christchurch it cannot be due to contaminated water, and this explanation will not account for the high incidence here and the low one in Auckland (11 per cent.).

(2) *Radio-activity of Water.*—Artesian waters are said to be highly radio-active. This might account for goitre in Christchurch, but radio-activity is lost by exposure and rippling. The water which supplies Timaru (South Canterbury) runs for a long distance in an open race, where radio-activity must be lost, but the incidence of goitre in Timaru school children is very high. Timaru is cited as representative of South Canterbury where the incidence is 62 per cent. Radio-activity of water does not explain the incidence of goitre in this district.

(3) *Salts Dissolved in Drinking Water.*—In many parts of New Zealand and in some parts of Canterbury, the only water-supply is stored rain-water, which contains no salts, but this does not prevent goitre. At Blackball, where only rain-water is used, 80 per cent. of the children showed enlarged thyroids. Lime has often been considered a cause of goitre, but in Kaikoura, the only limestone district in Canterbury, the incidence of goitre is rather lower than elsewhere. In any case, nearly all the water drunk is taken as tea, and therefore boiled, so that most of the lime-salts are precipitated.

Water-supply can have very little to do with New Zealand goitre: there is a record of ewes producing goitrous lambs when fed only on grass and turnips and getting no drinking water at all.

(4) *General Diet.*—There is evidence that excessive protein and also excessive fat in the diet may induce goitre. In New Zealand, however, the diet throughout the country is remarkably uniform, and there is a striking absence of local delicacies such as Scotch oatcake or Devonshire cream. There is no sufficient difference in the articles of food used in Auckland and Christchurch to account for the variations in goitre.

It is, however, of importance in this investigation that the bulk of the food consumed in any district in New Zealand is locally produced.

(5) *Geological Formation.*—This is of the first importance for the goitre problem in New Zealand, but in rather a special sense. In Europe, goitre is said to occur on carboniferous and silurian systems, but in New Zealand this generalization does not hold. Goitre-incidence depends on geology in the sense that it is determined by the presence or absence of certain salts in the soil, but their amount is too small to be of general geological interest. The presence of these salts in a given soil depends on the origin of the rocks from which it is derived, whether igneous or sedimentary, on the colloid nature of the soil which enables it to retain the salts against leaching, and on the surface disposition of the soil which determines its exposure to leaching and weathering.

(6) *Goitre as a Deficiency Disease.*—The geological characters of the soil just mentioned determine its content in iodine, and the whole of the evidence goes to show that the incidence of endemic goitre throughout New Zealand varies inversely with the amount of iodine in the soil, which is reflected in the iodine content of the food raised on it.

As mentioned above, some of his early field-work suggested to Hercus that the presence of iodine in the Lyttelton water might account for the reduced incidence of goitre there. Marine had arrived at and acted upon similar conclusions in America, and as long ago as 1853 Chatin had suggested that goitre was due to deficient iodine in air, food, and drink.

The main facts about iodine and the thyroid gland are briefly as follows:—Iodine was discovered by Courtois in 1812, and its distribution in rocks, water, air, and foodstuffs was very fully investigated by Chatin between 1850 and 1860. Many observers have added to the knowledge of the subject, which was summarized and extended by von Fellenberg between 1923 and 1925. He confirmed Chatin's findings in nearly all respects.

In 1896 Baumann found that iodine was a normal constituent of the mammalian thyroid, Oswald (1901) showed that it was present in the colloid. Marine and Lenhart (1909) found that the amount of iodine in the thyroid varied inversely with the degree of hyperplasia present. Marine and Rogoff (1916) demonstrated by perfusion experiments the great affinity of the thyroid for iodine. Kendall (1916) claimed to have isolated "thyroxin," the iodine-containing hormone of the thyroid. (Summarized from Hercus, Benson and Carter.)

It is evident, therefore, that there is a relationship between iodine and the thyroid gland, and that a deprivation of iodine is likely to induce changes in it.

Hercus has submitted the theory that endemic goitre in New Zealand is due to deficiency of iodine in food to a series of most exhaustive tests in association with W. N. Benson as geologist, and C. L. Carter and K. C. Roberts as chemists [1], [8]. The problem may be propounded thus: If iodine deficiency is a cause of endemic goitre it should be possible to demonstrate an inverse ratio between the incidence of goitre in different localities and the amount of iodine in the soils. Also a direct ratio should be maintained between the iodine content of the various soils and the vegetable and animal food grown upon it, since it is probable that men and animals depend on their food for their supply of iodine, though a shortage might be made good by a water-supply which happened to contain that element, as in the case of Lyttelton.

In this research the country was divided into thirty-three districts, soil-samples from each of them were assayed for iodine, and the local school-children were independently examined for goitre. Water, common salt and fertilizers were also analysed.

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Meantime an extensive geological research was carried out by Benson as to the distribution of iodine in soils, in relation to geological formation, the source of iodine in soils, and the incidence of goitre in relation to geographical conditions and to geological formation. The geological facts determine the amount of iodine in the soil, but the subject is too complex to be briefly summarized. A single instance may illustrate the conditions: There is a very high incidence of goitre in the Canterbury Plains. These occupy a broad stretch of country on the East Coast, and to the West is the high range of the Southern Alps, from which a number of great rivers cross the Plains. The soil is chiefly river gravel with very little colloid and very thoroughly leached. It is easy to understand that very little soluble substance derived from its original rocks is retained in it. The iodine in the soil is about four parts per 10^7, and the goitre incidence in the school children is about 60 per cent.

In all, some 500 different soils have been analysed. The method of chemical analysis is ignition of the soil with caustic soda, extraction of the iodide so formed with distilled water, and estimation of the iodide by a colorimetric method. A much more delicate technique was required for the estimation of iodine in foodstuffs, the one adopted is accurate to 0·5 microgramme (γ). Full details are given in the original papers.

A very striking inverse ratio was demonstrated between the incidence of goitre among school-children in any locality and the amount of iodine in the soil. Hercus, Benson and Carter state their results as follows: "The figures for goitre-incidence and soil-iodine have been plotted as ordinates and abscissæ respectively. . . . and a smoothed curve has been drawn through the points obtained. So closely do most of these points lie to the curve that it is apparent that an intimate connexion must exist between the amount of iodine in the soil and the incidence of goitre. Our curve indeed may be expressed approximately by the formula

$$y = \frac{360}{x} + 6$$

in which 'x' represents the amount of iodine in parts per 10^7 of the soil as determined by Carter's method and 'y' the percentage incidence of goitre in school-children, according to the general standard of diagnosis adopted in New Zealand." They add that for this to be perfectly true the iodine intake must be the sole factor involved, and it must be derived exclusively from the local soil, and great numbers of perfectly uniform examinations would be necessary; these conditions are not realized in practice, so that departures from the normal curve are inevitable, but the general relationship remains remarkably close.

The extreme instances and an intermediate one are the following:—

District	Children examined	Goitrous	Percentage	Soil iodine, parts per 10^7
South Canterbury ...	5,206 ...	3,228 ...	62 ...	3
Taranaki	5,493 ...	381 ...	7 ...	166
Wellington	2,633 ...	657 ...	25 ...	40

It is concluded therefore that the essential determining factor in the local incidence of endemic goitre in New Zealand is deficiency of iodine in the soil.

The proportion of iodine in the soil is reflected in the iodine content of the animal and vegetable foodstuffs raised upon it, which constitute the source from which human beings obtain their supply. This has been established in a further research by Hercus and Roberts [8], who have compared the iodine content of soils and their vegetables: all the common articles of food have been examined, and specimens from iodine-poor and iodine-rich soils have been compared. The iodine content of vegetables from the two kinds of soil sometimes varied as 2 to 3, sometimes

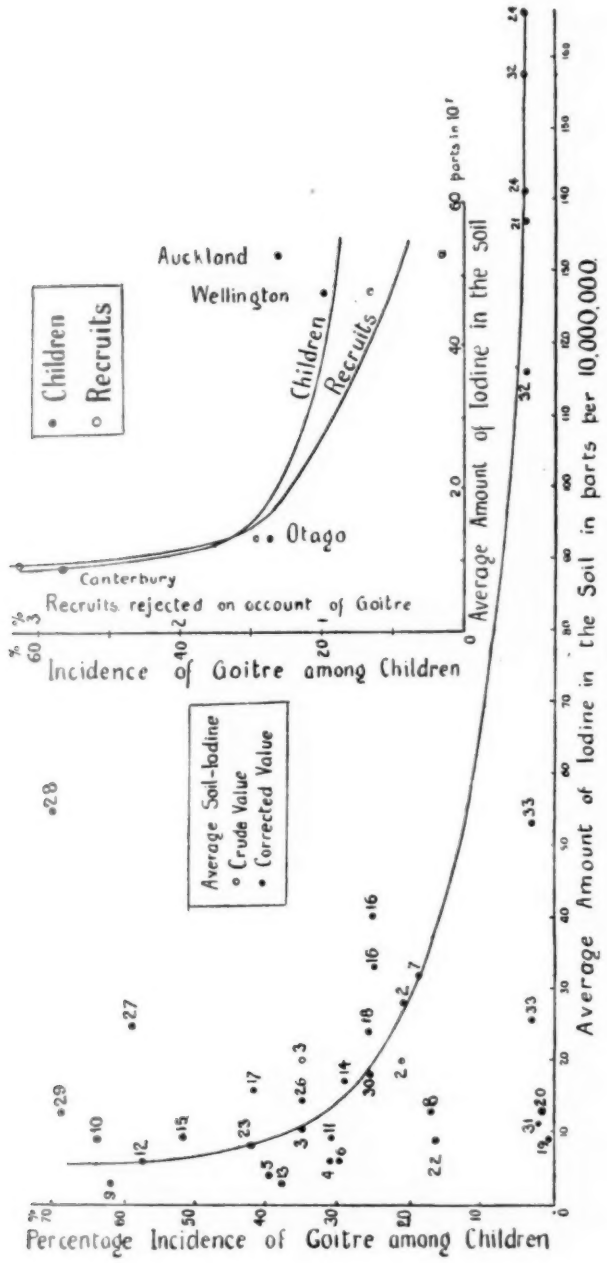

FIG. 1.

(From the *Journal of Hygiene*, 1925, xxiv, 3 and 4, by kind permission of the Editor.)

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as 1 to 2; a small number are here quoted. The iodine is estimated in microgrammes per kilo.

Iodine content of	Goitrous district	Non-goitrous district
Grass	47 γ per kilo	64 γ per kilo
Lettuce	12 ,,	24 ,,
Beef	9·5 ,,	16 ,,
Eggs	56 ,,	137 ,,

Incidentally interesting seasonal variations in the iodine content of vegetables were demonstrated, together with other important details which do not concern the present argument.

Finally the actual iodine intake per person per day was estimated for both a non-goitrous and a goitrous district. This was done for two separate fortnights for over forty people, half in each kind of district, with the following result:—

District	Mean iodine intake per person per day
Taranaki (non-goitrous)	35 microgrammes
Canterbury (goitrous)	20 microgrammes

Sea fish, especially shell-fish, are very rich in iodine; this explain why coastal Maoris who are large shell-fish eaters escape and have always escaped goitre, while the inland tribes suffer from it.

This research bears out the conclusion that iodine deficiency is the cause of endemic goitre in New Zealand: it also demonstrates the extremely minute quantities of iodine involved.

Prevention of Goitre in New Zealand.—If goitre is due to iodine deficiency it should be possible to prevent it by the ingestion of iodine. This is, of course, no new suggestion; Hercus found it to have been recommended by Prevost, of Geneva, in 1849, and to have been tried in Austria in 1855, and in Italy in 1859, but these attempts failed through overdosage. Marine and Lenhart (1917) gave about 3 gr. of sodium iodide for ten days in two terms to school-girls with excellent results, no normal girls became goitrous in thirty months.

In 1921, Hercus and Baker [10] gave from 40 gr. to 120 gr. per annum to school-children according to age, but the administrative difficulties turned out to be very great. The results were only fairly good, 39 per cent. of the treated, and 55 per cent. of the untreated became goitrous in the course of a year. The results among those already goitrous were rather better, but the method was clumsy and unsatisfactory. As the minute dose actually required became realized, it was felt that the necessary amount could be obtained in a more evenly distributed and natural manner by the use of iodized salt for kitchen and table purposes. By this means the whole population can be reached, not merely the school-children. Iodized salt, as authorized by the New Zealand Health Department, contains four parts per million of potassium iodide.

The whole success of the prophylaxis of endemic goitre by this means depends on the recognition of the infinitesimal dose of iodine required for physiological purposes, which may be considered on the following lines: Primitive marine invertebrates have a segmental excretory system which communicates direct with the body cavity, so that the body-fluid is identical with sea-water, which contains about 3·5 per cent. of salts, chiefly sodium chloride. Land mammals, developed from marine invertebrates, retain a body-fluid similar in saline composition to sea-water, diluted about five times, and containing about 0·73 per cent. of salts, chiefly sodium chloride. It is well known that sodium chloride derived originally from marine sources has to be artificially supplied to land-animals, especially man, and presumably the same is true of iodine, unless it is derived through plants from soil, chiefly soil of igneous origin. If the food supply comes from soil in which insufficient iodine is present, it must be

artificially supplied, like sodium chloride. The iodine content of sea-water is about 0·2 parts per 10^7, so that if the usual proportion is maintained, the iodine content of the body fluid is probably about 0·04 parts per 10^7. Ascertained figures are not available to the writer, but it is evident that only minute amounts are involved.

Iodine in soil and foodstuffs is calculated in parts per 10^7, and the average for soils in New Zealand is twenty-seven parts. Parts per 10^7 are about equivalent to grains to the half ton. The unit of measurement of iodine is the microgramme (γ), $\frac{1}{1000}$ part of a milligramme; there are about 60,000 γ in a grain.

The thyroid is exceptionally rich in iodine. Estimates of the amount present in the normal thyroid vary between 1 and 2 milligrammes per gramme of dried substance—the former is usually accepted—with a total content of 25 mgm. To maintain this, it is estimated that 10 mgm. of iodine (10,000 γ) are required per annum, or 200 γ per week, or 30 γ per day.

Iodine is in very common therapeutic use, and enormous doses are tolerated in non-goitrous districts without effect upon the thyroid; it is very rapidly excreted in the urine. But in goitrous districts it is of the first importance to keep the prophylactic dose limited to the minimum required for efficiency. The reasons for this are discussed under the head of "Toxic Goitre" below. It will be seen that the doses given by Marine and Lenhart, and also by Hercus and Baker in their early work are greatly in excess of these requirements. In young children this produces no bad results, but such excess is dangerous when given to the general public in goitrous districts; as will be shown later, there is considerable risk of inducing toxic goitre by so doing.

Hercus has conducted experiments to test the safety of iodized salt of the authorized strength (four parts per 10^6). It is estimated that 6 grm. of sodium chloride (6 million γ) are consumed by each person daily as common salt. If the salt contains four parts per million of iodide, the amount consumed daily is 24 γ, which is sufficient to make up the difference in the amounts consumed in goitrous Canterbury and non-goitrous Taranaki. To make sure that this was well within the limits of safety, the dose was doubled in a selected community. At a Dunedin orphanage common salt containing eight parts of potassium iodide per 10^6 was used, with the result that after eighteen months no normal children had become goitrous, and nearly 70 per cent. of the existing goitres showed a measurable decrease; no cases became toxic.

The use of iodized salt of a strength of 1 : 200,000 for two years in the Canton Appenzell am Rhein in Switzerland is said to have reduced the percentage of goitrous babies born from 50 per cent. to *nil*, and to have reduced the operations for goitre by 75 per cent.

The view is put forward that the only important cause of endemic goitre in New Zealand is deficiency of iodine in the diet, and that it can be effectively prevented by the use of iodized salt of a strength of four parts per 10^6. This view is not universally held, nor at present very fully acted upon, but it is the outcome of a very extensive and very careful research, the only one which has been attempted on this subject in the country.

Toxic Goitre.

Endemic goitre is thus shown to be highly prevalent in New Zealand; the incidence of toxic goitre is also considerable. The term "toxic goitre" is here used to include exophthalmic goitre, and refers to all cases which show tachycardia, tremor, and increase in the basal metabolic rate. Opinion is divided as to whether exophthalmic goitre should be put into a separate category, but there appear to be good grounds for the view that exophthalmos is an accidental symptom in toxic goitre, generally occurring in the hyperplastic, and also in the graver cases, but no

more essential to any particular disease of the thyroid than is ophthalmoplegia essential to the diagnosis of tabes. Nearly all cases of toxic goitre show at least a stare, of which the patient is often conscious, the whole of the iris being exposed when the eye is held open without effort, and the difference between this and actual protrusion of the eyeball appears only to be one of degree.

Incidence of toxic goitre.—The number of cases of thyroid disease admitted to the public hospitals in the ten years 1916-1925 was 3,083, with a fairly steady increase from year to year. In 1925 the number was 503, which was eighty-eight per thousand, nearly one case in eleven, of the total admissions. The number of deaths from thyroid disease during the decade was 524, of which 403 were due to exophthalmic goitre. The number of thyroid deaths occurring per annum has also shown a general increase; in 1925 the number was seventy-four, which is equivalent to 0·56 per thousand of the mean population. The figures for the incidence of toxic goitre and for deaths from thyroid disease are much higher in New Zealand than in Australia.

It has been argued that endemic goitre is due to deficient iodine, and an attempt is now made to estimate the relation of iodine to toxic goitre, from which a corollary is to be drawn as to the dose which it is safe for the general public to use for prophylactic purposes in goitrous districts. This is rendered possible by the work of Drennan [5], who has been a close student of the pathology of goitre in New Zealand for more than ten years.

Iodine and the Normal Thyroid.—The thyroid gland has, no doubt, many functions, but the only one capable of measurement is the maintenance of the basal metabolic rate. It is believed that this depends on the secretion of exactly controlled quantities of thyroxin, which contains iodine. The thyroid requires a certain fairly regular supply of iodine: if a greater amount than necessary is ingested the gland takes up so much as it requires and rejects the rest, which is rapidly excreted in the urine. No harm will come to the strictly normal thyroid from excess of iodine.

The thyroid, like all organs, has great powers of adaptation, and makes certain dispositions when its supply of iodine is inadequate. These have been widely discussed by pathologists, but Drennan is only able to recognize "two main phases, the resting, storing or colloid, and the active, secretory or hyperplastic." The thyroid may respond to deficient iodine by increasing the size and number of its alveoli, and filling these with colloid at a low concentration, or it may secrete actively without storage. A colloid goitre may show a high total iodine content, but a low content per gramme of substance.

Both these conditions are to be found in thyroids examined post-mortem which were not enlarged during life. They represent the normal response to moderately deficient iodine intake, for which these thyroids were able to compensate. The simple endemic goitres show these conditions on a scale large enough to produce naked-eye enlargement of the gland. Further, the same thyroid may show increased colloid in some parts and hyperplasia in others.

The simple goitre either stores every trace of iodine which comes in increased colloid areas, or elaborates and secretes thyroxin without any storage, and different parts of the gland may perform both these varieties of function. Hæmorrhage, cyst-formation, fibrosis and atrophy all occur in such thyroids, and parts of the glands may show isolated areas of increase, forming "adenomata." There is no clinical means of determining whether a given goitre is colloid or hyperplastic. The obvious treatment is to give iodine to such patients. In early stages of goitre, such as those in school children, relatively large doses may be given without risk, the yearly requirement being estimated at 10,000 γ; Hercus and Baker in their early work gave 7,000,000 γ, without bad results, and one school doctor gave 18,000,000 γ,

also without harm. Presumably the young thyroid is able to readapt itself to the excessive supply and to reject what is not required.

If this were the case throughout life no precautions would be needed in goitrous districts and ordinary therapeutic doses of iodine could be given without risk, but it is not so.

In practice, if iodine in excess is given to an adult with a thyroid long adapted to a low iodine intake, toxicity is often induced, hence minimal doses only are permissible in salt designed for general use. It appears that under various conditions of stress there is a demand for increased thyroid secretion ; the simplest instances are the sexual stimuli in women : even the normal thyroid enlarges, albeit very slightly, in menstruation and in pregnancy, and it is under such stresses that simple goitres become toxic. Drennan has shown that the colloid goitre often contains a large total amount of iodine, and it may be that under the stress a " mass effect " is produced, with excess of thyroxin secreted. Young adult students, under the stress of examinations, are also liable to suffer from toxic symptoms.

In a potentially toxic goitre of this kind the thyroid either stores all available iodine or uses it for immediate internal secretion, and if excess of iodine is given, excessive secretion frequently results, with precipitation of toxic symptoms. The following are instances :—

(1) A woman medical student, holding the highest place in her year, and doubtless working at very high pressure, ill-advisedly took 15 gr. of potassium iodide three times a day for her goitre, which was becoming large enough to be a disfigurement. All the signs of Graves' disease rapidly developed, and operative treatment was ultimately required.

(2) A male medical student, aged 23, had a large soft goitre, for which he took, on his own responsibility, 200 gr. of potassium iodide in nine months, rather less than a grain a day. At the end of that time he had lost a stone of weight, and had exophthalmos, tachycardia, tremors and increased basal metabolic rate, but his goitre had quite disappeared. He recovered in a year without operation. In this view the thyroid, hitherto accustomed to a low iodine intake, and perhaps called upon by the stress of examinations to increased action, was led into hypersecretion by the iodine excess of about 40,000 γ a day.

But toxic symptoms may be induced in the susceptible by very much smaller doses than these. Dr. Frank Fitchett has kindly allowed the use of the following cases from his practice :—

(3) A patient consulted him for obesity, which concealed a goitre. Potassium iodide was prescribed as an adjuvant to other treatment. The goitre became toxic and the patient was laid aside for months with intense symptoms of hyperthyroidism.

(4) A patient with goitre consulted him for pleural effusion. The chest was painted with tincture of iodine, after which the goitre became toxic.

Finally, at a ladies' college, iodized salt was used on an American formula which contained 1 part in 5,000 of potassium iodide. This is fifty times as strong as the salt now authorized by the New Zealand Health Department, though it only implied the consumption of the not very formidable dose of $\frac{1}{1000}$ grain of iodide, or about 60 γ a day ; but after its introduction three of the students consulted Dr. Fitchett for symptoms of hyperthyroidism.

Dr. Fitchett pertinently asks how far the frequent prescription of iodides in ordinary therapeutic doses, for bronchitis or rheumatism, may be responsible for the high incidence of toxic symptoms in goitrous patients in New Zealand. Whatever other causes of hyperthyroidism there may be, and there is no pretence that iodine excess is the only one, it will be seen that iodine is no drug to play with in goitrous communities, and that when exhibited in salt for general prophylactic use, minimal

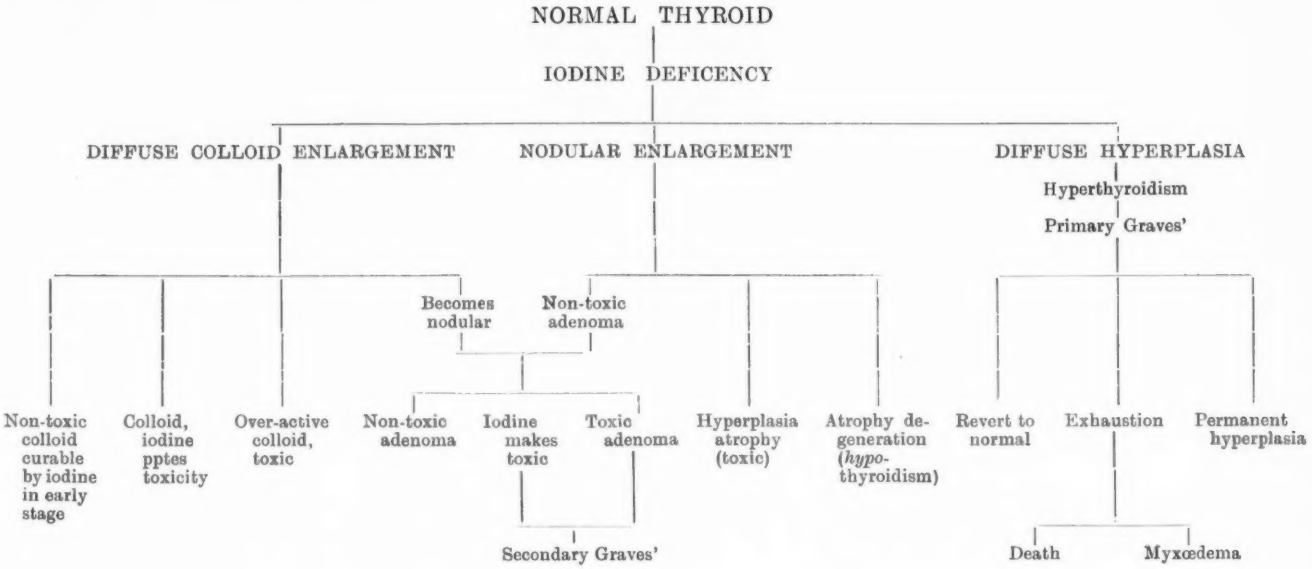
NORMAL THYROID
IODINE DEFICENCY
DIFFUSE COLLOID ENLARGEMENT
NODULAR ENLARGEMENT
DIFFUSE HYPERPLASIA
Hyperthyroidism
Primary Graves'
Becomes nodular
Non-toxic adenoma
Non-toxic colloid curable by iodine in early stage
Colloid, iodine pptes toxicity
Over-active colloid, toxic
Non-toxic adenoma
Iodine makes toxic
Toxic adenoma
Hyperplasia atrophy (toxic)
Atrophy degeneration (hypo-thyroidism)
Revert to normal
Exhaustion
Permanent hyperplasia
Secondary Graves'
Death
Myxœdema

FIG. 2.

doses only are permissible. A corollary to this is that proprietary goitre remedies, all of which contain iodine, should on no account be employed, and their use should properly be prohibited.

Drennan has devised a schema to show the apparent influence of iodine on the thyroid, of which the elementary points are indicated in fig. 2.

It demonstrates the following points: The normal thyroid, exposed to a deficiency of iodine, displays one of three things: (1) Diffuse colloid enlargement; (2) nodular enlargement (adenoma)—the enlargement may be either colloid or hyperplastic or a combination of the two; (3) diffuse hyperplasia.

(1) *The Diffuse Colloid Enlargement.*—This often remains simple; it is curable with iodine. Probably most of the goitres in children are of this kind, in whom overdosing does not matter, but for adults minimal doses are required. Such a goitre, successfully treated with iodine, is reduced unless this is prevented by cystic or other changes; it reduces its storage area, not so much now being needed, and adapts itself to the increased intake.

If a patient with such a colloid goitre be subjected to stress, as from pregnancy, the mass effect may be induced; the large store of iodine, albeit at low pressure, may produce hormone in excess, and toxic symptoms may result. Iodine excess may readily precipitate toxicity.

It is true that this is not a complete explanation of the metamorphosis of simple into toxic goitre. Every case of simple goitre exposed to stress does not become toxic. The high incidence of goitre at the Christchurch Mental Hospital has been mentioned, but it is said that toxic goitre is practically unknown there. Dr. Hercus in a private communication stated that he could only find three cases with toxic symptoms in their records, although the patients were lunatics, often subject to all kinds of mental and physical stress.

The goitre, originally simple colloid, may become nodular, and is then indistinguishable from the thyroid which first responds to iodine deficiency by becoming nodular; "simple adenoma" includes both conditions.

(2) *The Simple Adenoma.*—This may remain non-toxic and give no more trouble than the simple colloid. On the other hand it is clinical experience that the nodular kind is more likely to become toxic and form the "toxic adenoma." Iodine in excess may induce this change, and this is the type most liable to become toxic, even to the extent of exophthalmos, under stress. These are those who have had goitre for years without symptoms, but who go down suddenly with secondary Graves' disease. This is particularly likely to occur about the menopause: Drennan suggests that such thyroids are adjusted to a low iodine intake, and when the increase of hormone secreted at the menstrual period is no longer required, the thyroid is overcharged for its needs, and toxic symptoms result. This may occur whether the enlargement is colloid or hyperplastic.

In some cases the adenomata atrophy and any degree of hypothyroidism may result.

(3) *Diffuse Hyperplasia.*—When the thyroid responds to iodine deficiency in this way, all the iodine received is secreted immediately in the hormone without storage. These are the cases of manifest, rapidly developed hyperthyroidism, "primary Graves' disease," who had no goitre at all till it suddenly appeared with toxic symptoms. Admittedly, their association with previous iodine deficiency is less evident.

These are the cases which are benefited by Lugol's solution; how this happens is not quite clear. It seems that the thyroid has run on a very short ration of iodine, all of which has been used for hormone, and given this excess of iodine, it is able to perform the function of storage, which gives it a short period of quietude, during which surgical treatment is possible.

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Such cases occasionally subside and revert to normal with rest and physiological doses of iodine; probably a greater number continue hyperthyroid invalids for the rest of their lives, unless operated upon; some of them become myxœdematous. Some of them become rapidly exhausted and die, and show no power to respond to treatment with Lugol's solution.

A case showing complete exhaustion was the following:—

A man of middle age was admitted with rapidly developed Graves' disease, but not very severe symptoms. He was treated with rest and the empirical drugs in common use, with slight temporary improvement, but he soon became very seriously ill, he was light-headed, extremely restless and difficult to control, and he rapidly wasted. Lugol's solution was then prescribed, though not in doses as large as those advised by Plummer, but the condition grew much worse. The surgeon who was coöperating over the case thought it imperative to operate at this point, and some of the thyroid arteries were tied, but the patient died almost immediately. Post-mortem, a completely exhausted gland was found, probably quite incapable of responding to any stimulus. In this case it is just possible that thyroxin might have supplied the needs of the body sufficiently to have allowed the thyroid an opportunity to recover.

It appears to observers in New Zealand, at any rate to those who have followed the work of Hercus and Drennan sympathetically, that endemic goitre is induced by iodine deficiency, though it is not claimed that this is the only cause of thyroid enlargement. Since that gland controls the metabolic rate, it must often be called upon to act to excess, and is then as likely to enlarge as any other over-active tissue. As a gland of internal secretion it has evident—though little understood—relations with the other endocrine organs, and it enlarges at puberty, during menstruation, and in pregnancy. It often enlarges during infections; doubtless McCarrison's Indian cases are of this character, and a goitre is often reduced by the removal of a septic focus. But in New Zealand, iodine deficiency is the most important predisposing cause of all forms of goitre, and the infinitesimal dose involved has been emphasized above. Drennan's scheme shows how excess of iodine may precipitate toxic symptoms in a goitre adapted to a low intake of that substance.

These researches are far from solving the problem of toxic goitre, but after their experiences in endemic goitre, the first contribution for New Zealand workers to make to the problem of toxicity is the investigation of the iodine problem therein. Whatever else may be involved, it is submitted that the work here summarized indicates that iodine has at least an important bearing on the occurrence of toxic symptoms.

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Section of Ophthalmology.

President—Mr. Ernest Clarke, C.V.O., F.R.C.S.

(Clinical Meeting held at St. Thomas's Hospital, March 9, 1928.)

CASES.

Asymmetry of Face.

By P. G. Doyne, F.R.C.S.

W. W., aged 11. Facial asymmetry since birth. No diplopia. Vision: Right, $\frac{6}{12}$; left, $\frac{6}{6}$.

X-ray examination shows dense bony tumour in right frontal region.

This case was shown at a meeting of the Section for the Study of Disease in Children. It is suggested that it may be a case of plagiocephaly. The right eye is more hypermetropic than the left, suggesting a smaller orbit. In other respects the boy is normal.

Case for Diagnosis. ? Parinaud's Conjunctivitis.

By P. G. Doyne, F.R.C.S.

T. F., aged 11. November 28, 1927, was struck on right jaw (in a fight). The eye swelled up the next day, as did also the pre-auricular gland on the same side. There was some chemosis, and a slough on the fold of the lower fornix.

X-ray and bacteriological examinations, negative. The last week the pre-auricular gland became soft, and pus was aspirated from it, cultures of which were sterile. Local treatment has not caused any change, but the case shows some improvement.

Mr. E. R. Chambers said that he had had a case similar to this and of some months' duration. The glands were much swollen but the bacteriological culture was negative. A section was reported to be granulation tissue. The only treatment was a 2 per cent. boric acid lotion and general irradiations with ultra-violet light. The condition entirely cleared up in about two months. The child's general health was considerably improved by the light treatment.

Fleshy Mass on Right Cornea.

By H. M. Joseph, M.C., M.B. (for Montague Hine, F.R.C.S.).

Patient, male, aged 84, had six years ago, an ulcer at the upper edge of the cornea, with pannus and slight entropion; two years ago a small, fleshy mass appeared at site of scar. This mass steadily increased until it reached its present size.

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On Examination: A fleshy mass, white in colour, covers the upper part of the cornea to which it is fixed. There is no infiltration of the cornea beyond the margin of the growth and no apparent involvement of the sclerotic.

Discussion.—Mr. ERNEST CLARKE (President) said that the mass appeared to be growing rather deeply into the cornea. He advised the excision of a small portion for microscopical examination.

Mr. A. F. MACCALLAN said that he had seen a number of similar cases in Egypt in patients who had healed ulcers of the cornea, and he had always looked upon the condition as a granuloma which had become somewhat fibrosed. It did not change at all, and no treatment was of any use.

Mr. H. NEAME said that Mr. Foster Moore had shown a similar condition, but with a rather more definite margin, in a girl aged about 14. In Mr. Moore's case the opacity did not fade into a haze, as in this case, but no diagnosis could be arrived at.

Tumour of Iris.

By A. F. MACCALLAN, C.B.E., F.R.C.S.

A. E., AGED 36, male.

I saw this case a few days previously, and have brought it for diagnosis. The condition was first noticed about eleven years ago, but the patient cannot be certain that it has not always been present. He is slightly myopic, and the reason why he came to hospital was because he does not see so well in the distance as he did formerly. Vision: Right, with $-0{\cdot}75$ sph. $= \frac{6}{6}$. Left, with $-1{\cdot}25$ sph. $= \frac{6}{6}$. The fundi are normal. As seen with the slit lamp, the tissue of the decolorized tumefied area, in which the fibres of the iris are distinctly seen, seem to merge gradually into the normal pigmented iris tissue. I personally think that the tumour is a nævoid growth of the iris.

Discussion.—Mr. H. NEAME agreed with Mr. MacCallan's suggestion that the tumour was a nævoid growth, or a melanoma, related to the pigmented growths in the skin, sometimes called nævi, but not to the vascular nævi.[1] It should be measured accurately by the corneal microscope in both dimensions, and watched for a few months for any increase in size, which would, he thought, indicate an operation. He believed that it was growing and would ultimately prove to be malignant. He was not sure whether an iridectomy would remove it.

Mr. C. H. WALKER said that about ten years ago he had had a patient, a boy, aged 16, who had a small tumour of the iris, the same in appearance as in this case. Some minute vessels had been just visible in the growth. It had increased slowly, so he performed an iridectomy, and believed he had removed the whole of the tumour, as, after a month or two, there was no sign of it. A section of the removed portion was examined by Professor Walker Hall and reported to be a sarcoma, with scarcely any pigmentation. After about two years, however, there had been distinct recurrence running along in the angle of the anterior chamber and evidently tending to spread. He had sent the boy to Mr. Treacher Collins, who said that undoubtedly there was the clinical appearance of a sarcoma. The eye was excised seven years ago and there has been no recurrence.

[1] Nævus pigmentosus (pigmented mole).

Hypertrophic Scars in Retina.

By ARTHUR GRIFFITH, F.R.C.S.

H. R. J., AGED 33, male.

History.—Seven years ago vision of the right eye failed for over a month. There is a history of several injuries to that eye, but none shortly before the failure of vision.

In 1923 the patient had pleuritis, and three months later hæmoptysis, and since then he has had sanatorium treatment for pulmonary tuberculosis.

On examination three long curving lines of scar tissue are seen in the retina, raising the vessels where they pass under them. The retina is detached downwards

Hypertrophic Scars in Retina.

and outwards. (The retinal detachment is actually more extensive than it appears to be in the illustration.)

One or two questions arise in this case. The scar tissue shows no relation to the arrangement of the retinal vessels. One possibility is that the condition began with hæmorrhages from ruptured choroidal vessels; that fibroblasts from these vessels, joining up, formed the lines of scar tissue and the scars, by contraction, pulled off the retina. Another possibility is that the patient had first of all a traumatic detachment of the retina, and that these scars were the result of retinal injury.

Scarring of the Nerve Head.

By ARTHUR GRIFFITH, F.R.C.S.

A. M., A BOY, aged 8.

Left eye found to be defective recently. Patient is the fourth child of a family of six. Parturition was natural and easy. There is no history of injury.

Vision: Right $\frac{6}{9}$; left $\frac{6}{36}$. Astigmatic.

Scarring of the Nerve Head.

In the left eye there are masses of scar tissue around the optic disc. The picture is that of partial avulsion of the optic nerve, possibly a birth injury. There are all kinds and degrees of lesions following birth injuries, from severe ones such as that shown by the exhibitor four years ago[1], to the slight cases like one seen a

1 *Proceedings*, 1924, xvii (Sect. Ophth.), 23.

few days ago, in which, despite a clear history of forceps injury, there appeared to be nothing previously wrong except high astigmatism, and the fundus seemed to be normal. It is possible to get this degree of birth injury in ordinary delivery without forceps.

Mr. ERNEST CLARKE (President) said that the optic nerve might be injured in ordinary delivery without forceps, considering the distorted condition in which the head was sometimes delivered, even when no instruments were used.

Fundus Pictures.

By ARTHUR GRIFFITH, F.R.C.S.

(I) RIGHT eye, gumma of sclera. (II) Left eye, gummatous scleritis. (III) Rash on forearm.

Patient, male, aged 49.

History.—Twenty-five years' service in Army, ending in December, 1919. Right eye painful and red for three months. Left eye swollen for two months.

Vision: Right $\frac{6}{9}$, N.I., H.M. + 0·75 sph.; Left $\frac{6}{9}$, with + 1·25 sph. = $\frac{6}{6}$. Right eye, gummatous ulcer of sclera. Rectus externus destroyed, and eye convergent. Patch of acute choroiditis downwards and outwards. Vitreous haze. Left eye, gummatous scleritis, temporal side of limbus. Wassermann reaction strongly positive.

After three weeks' treatment, this patient regarded himself as sufficiently improved, and declined further treatment.

Aniridia with Dislocated Lens.

By A. HUGH THOMPSON, M.D.

THE patient, a girl aged 12, is a pupil of the Swiss Cottage Blind School. The school nurse says that she has seen the child's father, who is affected in just the same way. Each lens is dislocated upwards. Vision is $\frac{4}{60}$, with a + 6 lens, so that if the lens were in place the child would be myopic. No iris is to be seen. There is vertical and rotatory nystagmus.

Senile Central Exudative Retinitis Simulating a Second Optic Disc.

By A. HUGH THOMPSON, M.D.

PATIENT, male, aged 67.

First seen February, 1927, when the sight of the right eye had been getting dim for three or four months. It was then reduced to "fingers at 2 ft.," and has remained so ever since.

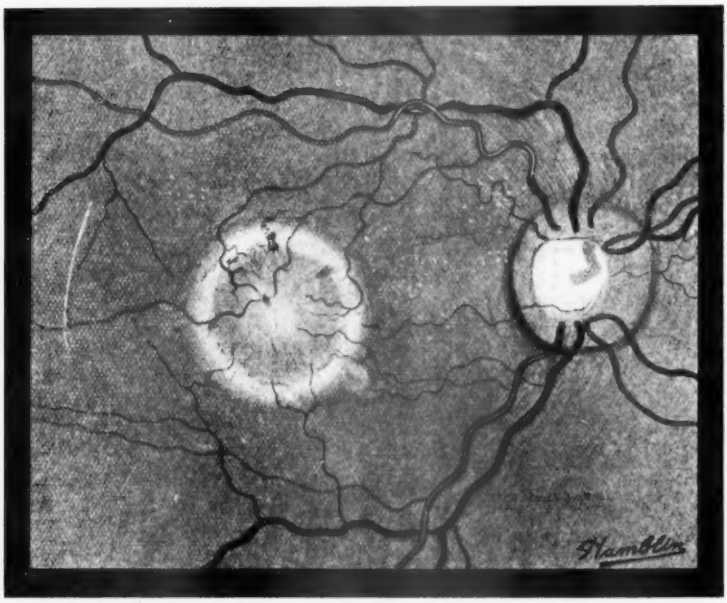
The left eye had vision $\frac{6}{6}$ and was normal in appearance, except for some pale white spots between disc and macula. Vision was noticed to be worse about November, 1927. In January, 1928, it was $\frac{6}{9}$, and in February, $\frac{6}{18}$.

52 Thompson: *Senile Central Exudative Retinitis*

On Examination.—Right macula: Disc-like degeneration with very little pigment and normal retinal vessels superficial to it.

Left macula: Early stage of grey degeneration. This was not present a year ago. Some evidence of retinal arterio-sclerosis at crossings of vessels.

Right: Large absolute central scotoma. Left: Central scotoma for red and green.



Exudative Retinitis simulating a Second Optic Disc.

Discussion.—Mr. HUGH THOMPSON said that he considered the condition in the left eye to be an early stage of the condition seen in the right eye. He had called this a case of choroiditis, but it might be described as macular degeneration. What the pathological condition was he would like to know, and still more, whether any treatment was of use. Three teeth had been extracted and no other septic focus could be discovered. The patient had been a somewhat heavy smoker, but was now, under orders, smoking less.

Mr. H. NEAME said he had an impression that there was a swelling on the right macula, and he felt certain there was a small hæmorrhage downwards and outwards just at its edge. It looked to him like one of those central senile exudative retinitis cases on which Mr. Devonport had read a paper recently.

Mr. R. C. DAVENPORT said that from the description and the picture, he had no doubt that this case came into the class of senile macular exudative retinitis; it showed vascular degeneration rather than sclerosis. The series of cases he had reported did not exhibit increased blood-pressure. The second eye became involved in most cases; small spots appeared first and then there was the sudden onset of a fairly large, whitish, definitely raised mass.

Loss of Upper Half of Visual Field in One Eye. ? Cause.

By G. W. KENDALL, M.D.

THIS patient is a woman, aged 22, who came to me in December of last year complaining that she could not see the upper half of anything with her left eye. She noticed this condition coming on about four years ago, and it has been as at present for two years. The right vision is $\frac{6}{6}$, and the left $\frac{6}{6}$. There is complete loss of the upper half of the visual field in the left eye. The lower half of the disc is pale, and there is a slightly shaded area at the macula with some white spots. There appears to be an obstruction of the inferior retinal artery. The complete loss of the upper half of the field is interesting. Her doctor reported that none of the usual causes of embolism was present and that she was apparently perfectly healthy.

Changes in Macula. ? Familial Macular Degeneration.

By HUMPHREY NEAME, F.R.C.S.

PATIENT, a girl, aged $7\frac{1}{2}$

1926. Defective sight noticed through patient holding book close to eyes. (Eight months child. Always backward.)

Family History.—Only child. Mother had no miscarriages. Mother's paternal aunt, aged 50, is quite blind, with defective vision from childhood.

February 9, 1927: Pupils equal, active.

Vision: Right $\frac{1}{24}$, retinoscopy correction about $+\frac{1{\cdot}0}{+1{\cdot}0}$ 90°

Left $\frac{1}{60}$, " " " $+\frac{1{\cdot}0}{+1{\cdot}0}$ 180°

Right and left macular pigmentation, ? familial degeneration. Wassermann reaction positive. The frontal and parietal eminences are prominent. Teeth not suggestive of congenital syphilis.

February, 1928.—Right macula: fovea pale. Ring of coarser peppering around fovea. Finer scattered peppering in macula around this. Left macula: Similar, but less well-marked changes. Mental condition satisfactory.

The only uncertainty in my mind is as to whether the condition might possibly be a mild congenital syphilitic lesion. There is a central pale area right in the fovea; and around it, peppering the background, is a ring of dots.

Optic Atrophy.

By M. WHITING, O.B.E., F.R.C.S.

PATIENT, aged 10, has had "fits" from the age of 6 months to that of 7 years. His vision was good until November, 1926, since when it has failed. Vision now with glasses, right $\frac{6}{12}$, left $\frac{6}{18}$. No family history of defective sight. Wassermann negative. X-ray negative as to pituitary region. Fields show slight general contraction. What is the cause of the optic atrophy and of the faint peppery pigmentation around discs?

54 Wilkinson: *Retinal Disease with Massive Exudation*

Retinal Disease with Massive Exudation.

By P. B. WILKINSON, M.B., B.S.

PATIENT, a girl, aged 16.

At the end of 1925 the vision of the right eye began to fail, and by November, 1927, was down to $\frac{6}{24}$. The left eye remained normal until August, 1927, when it became "clouded." The vision in the left eye failed rapidly until, by November, 1927, it had been reduced to inaccurate finger counting in the lower half of the field. During October, 1927, there were "moving balls of light" and a "diffuse moving glow" constantly in the field of vision. These phenomena were not definitely localized in the field, nor were there any higher-grade visual hallucinations. From September to November, 1927, she had occasional right fronto-temporal headaches which were unaccompanied by nausea or vomiting. There were no definite signs elsewhere. The family history is negative from all points. The main eye changes were the presence around both discs of extensive white areas extending fairly far forwards. Both the retinal arteries and veins were enormously distended, but neither showed pulsation. There were no hæmorrhages.

Points of Special Interest.—(1) Exploration in right occipital region by Sir Percy Sargent, December 8, 1927: no sign of tumour was found, but there was a gross excess of fluid in the subarachnoid space. (2) The enormous dilatation of the retinal veins, and the fact that the retinal arteries are almost as large. (3) The complete absence of signs elsewhere. (4) The bilaterality of the eye signs.

Section of Neurology.

President—Dr. Edwin Bramwell.

[*March* 8, 1928.]

DISCUSSION ON NARCOLEPSY.

Dr. Kinnier Wilson.

My interest in this subject dates from 1907 when I was house physician for Sir William Gowers, who published in that year his book, "The Borderland of Epilepsy," in which was a chapter on "Disorders of Sleep," with a clear description of narcolepsy. No attention, however, has been paid to this work by recent writers.

I have been on the look-out for narcolepsy since 1907, and with one or two minor exceptions have never seen an adequate case of it until 1927. Then I saw no fewer than four cases within a few months. Since then I have seen three more. Spiller, in an article on the subject in 1926 (*Journal of the American Medical Association*), said: "A few years ago it was exceptional for a neurologist, even with a large experience, to see a case of typical narcolepsy. I have been impressed by the fact that at least three cases have come under my observation within a year." We may therefore suppose that the condition has come into prominence, either because there is an increase of cases, or because our interest in the subject has been awakened by epidemic encephalitis. As a matter of fact some of my cases date from long before the outbreak of epidemic encephalitis in 1918. During the last few years a few cases of the syndrome have been described as following epidemic encephalitis, but very many other cases have nothing to do with that affection, as far as can be ascertained.

In no circumstances, in my opinion, can narcolepsy be regarded as a disease; it can be no more than a symptom. To call narcolepsy a "morbus sui generis" is nosologically erroneous. If narcolepsy appears—as it does occasionally—in, and as a sequel to epidemic encephalitis, it cannot be regarded as other than a symptom or a syndrome. I therefore prefer to speak of narcolepsies, in the plural, meaning thereby all those types of symptoms or combinations of symptoms—symptom-complexes—in which there is a disorder of the function of sleep, in the direction of hypersomnia.

"Narcolepsy" is not, perhaps, an ideal word. The word *νάρκη* according to Liddell and Scott signifies "numbness, deadness (Lat. torpor) caused by palsy, frost, fright, etc.," and that is not quite the idea conveyed by narcolepsy. It has been suggested that we should use the word "hypnolepsy," a falling into sleep, a sleep ailment, and Sir William Gowers years ago suggested "somnosis," a term now quite forgotten.

The first patient I saw was a youth who, for two and a half years, had suffered from irresistible attacks of sleep, which came on any time and anywhere. Since their onset he has slept daily, sometimes many times a day. In addition to these attacks he has another variety—known since the original description of Gélineau in the early eighties. Gélineau described cases of narcolepsy in which, in addition to the sleeping, the patient suffered from peculiar attacks in which, under the influence of emotion, his knees gave way and he sank powerless to the ground. This has been called by subsequent writers "cataplexy."

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The second case is that of a young man who has had the symptoms for nineteen months. He differs from the first in that his first symptom was the cataplexy. He went in for a football sweep and won £5. At the moment of excited rejoicing his knees gave way and he fell down. From that day he has had repeated cataplectic attacks under the influence of emotion. He has also developed in characteristic form the attacks of sleep. A significant feature of this second case is that if the patient tries to avoid an attack of sleep the cataplectic attack will develop. I had the good fortune to examine him during a cataplectic attack, which lasted about ninety seconds, and found the knee-jerks completely abolished and an extensor response present. Two minutes after, his knee-jerks were extremely active, and the plantar response flexor. In other words we have here a phenomenon consisting in so complete an atonia, and outfall of reflective activity so fulminating that a man loses his knee-jerks and gets them back in two minutes, while his plantar response becomes extensor and flexor again in the same time. The patient was conscious throughout and described what was done.

The third case is that of an older man, who has had his symptoms since after the Boer war. His symptoms are identical with those of the other two, but there is an additional point of interest in his case. I attach the utmost importance to this: that in addition to the "sleep" attacks (we shall see in a moment that "sleep" is not a good word for them) and the cataplectic attacks, he has attacks of what can only be described as catalepsy or trance. When he is lying or sitting quietly in a chair he will suddenly find himself unable to move a single joint through the fraction of a millimetre. He will lie like that, to all appearances normal, his eyes shut, and yet, as he said to me, "somebody could come in and murder me and I could not move." He has burned his fingers by being incapable of letting drop the cigarette he held when the trance came on. This is a remarkably interesting condition, linking narcolepsy with phenomena with which superficially it does not appear to have much association.

It has been known for years that lesions in certain parts of the brain are associated with disorders of the function of sleep. A number of years ago Righetti collected 775 cases of verified cerebral tumour, in which 115 were noteworthy for pathological sleep. He compiled the following figures:—

Site of tumour	Percentage of cases showing abnormal sleep
Thalamus and third ventricle	34·7
Medulla, and pons	27·9
Hypophysis and vicinity	26·5
Corpora quadrigemina and epiphysis	25·6
Central gyri	10·0
Frontal gyri	6·0

Tumours in the vicinity of the third ventricle, hypophysis, epiphysis and corpora quadrigemina together form a far higher percentage of cases of pathological sleep than those of any other part of the brain, and the association is impressive.

In respect of narcolepsy, however, "sleep" is not a very accurate term; in a large number of cases the patient has not in fact been asleep, but has been awake, and aware of what is going on round about him, presenting only the appearance of sleep. It is obvious that we cannot consider a person asleep in a normal or physiological sense if he is aware of what is going on.

In a considerable number of cases, further, the patient is aware of what is going on round about him but *he is unable to move*. Not only so, but there are transitions and substitutions between the two; for instance, one of my patients, if he tried to prevent himself from sleeping, had a cataplectic attack. Gélineau, who described

narcolepsy forty years ago, found that his patient fell asleep under the influence of emotion, instead of having a cataplectic attack. The more excited he got the more irresistible was the desire for sleep. Here was emotion precipitating a sleep attack instead of a cataplectic attack. The cataplectic attack may come on without emotion, and other combinations are possible. We may thus regard the phenomena in many instances as a transition between sleep and catalepsy. By widening the conception of the condition in that way I think it is calculated to throw more light upon the peculiar types of case which do not fit into any strictly delimited category of narcolepsy.

With regard to the relation between narcolepsy and epilepsy. It has been argued by Dr. Adie that these are absolutely distinct from each other. Unfortunately, as I think, he calls them both diseases; I do not regard either as such. I think, moreover, that the resemblances are greater than the differences, and that we have here the possibility of a pair of corresponding opposites, in a Jacksonian sense. The patient who has a major epileptic attack is unconscious and his limbs are in motion. That is to say, his cortex is clouded, his stream of consciousness is interrupted, and his limbs are in tonic or clonic spasm. In the cataplectic attack the reverse occurs, because the patient is awake, knows everything that is going on, and yet is immobile. A fair number of cases are on record in which epilepsy and narcolepsy have been associated. I mentioned a short time ago, in a paper on epileptic variants, a case of epilepsy changing into narcolepsy. A man, aged 21, had had six epileptic fits between the ages of 2 and 7, and three or more between the ages of 9 and 14. These appear to have been characteristic major epilepsy. Since the age of 14 he has had no more epileptic attacks. Recently, however, for some months he has suffered from a different kind of attack, in which he loses himself, turns white, falls, or sits still unable to move. These attacks are brought on by uproarious laughter. It is undesirable that we should differentiate epilepsy and narcolepsy as if they were fundamentally distinct. My view is this: that contrasts between narcolepsies and epilepsies are merely contrasts between the symptoms, and it is perfectly possible that there may be analogous physiological mechanisms between them. If that is so, they should be allied instead of being dissociated.

If we agree that many narcoleptic cases are really trance-like states, catalepsy, immobility with awareness, then we must allow that these are phenomena of inhibition. Now some of the phenomena of ordinary epilepsy are inhibitory and not excitatory. A petit mal patient becomes immobile, strikes an attitude, drops what is in his hand, stares; he is inhibited, immobile. The aura of many cases of epilepsy is inhibitory. The phenomena, so far from being phenomena of excitation, are phenomena of inhibition, a fact frequently lost sight of in discussions on the epilepsies. It seems to me that in narcolepsies we have the same condition carried further, namely, that practically all the phenomena are phenomena of inhibition.

Dr. C. Worster-Drought.

In a paper to which Dr. Kinnier Wilson has referred,[1] which I read some years ago before the British Psychological Society,[2] I dealt mainly with "symptomatic" narcolepsy, as at that time I had not seen a case of "idiopathic" narcolepsy. Hence, I can entirely confirm Dr. Wilson's statement that it is only recently that such cases have been seen. Only within the last three or four years have I seen a true or idiopathic narcolepsy; that is, not a "symptomatic" narcolepsy depending upon some recognizable lesion or disease, but one with no physical signs of an organic condition.

1 "The Narcolepsies," *Brain*, 1928, li, 63.

2 *Brit Journ. Med. Psych.*, 1923, iii, 267.

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At present I have four cases of true narcolepsy under observation, three in men and one in a woman. This last case is unusual, as most of the cases occur in men. Dr. Wilson mentioned that in one of his patients the cataplectic attacks developed before the spontaneous sleep; this, I believe, is rather exceptional. In the cases I have observed the sleeping attacks have preceded the onset of cataplexy by periods varying from a few months to two years. Then, again, I can entirely confirm his observations on the extreme muscular hypotonia that occurs during the actual attack of cataplexy. I was fortunate enough to witness an attack in one of my cases. I asked a friend with a fund of humorous stories to relate one; the patient laughed heartily, and this produced an attack. I had little time to examine him as the attack was short; I was impressed most by the extreme manner in which the head fell forward and the lower jaw fell with a few clonic movements before it dropped completely. The arm- and knee-jerks were entirely absent during the attack. The patient was evidently quite conscious during the whole of the cataplectic stage, as afterwards he told me all that had happened and in what way I had examined him.

I entirely agree that, rather than a disease *sui generis*, one should regard true narcolepsy as a symptom-complex indicative of an underlying nervous disturbance, probably of an organic nature. We have yet to discover the nature and site of the lesion, and I think the evidence in favour of a hypothalamic localization is suggestive, but far from conclusive. An interesting observation for comparison is that in a recent patient with the clinical signs of syringomyelia, my house physician (Dr. Allen) pointed out that while he was taking the history of the case the patient repeatedly fell asleep. It was learnt that he was quite accustomed to fall asleep spontaneously during the daytime. We inquired for symptoms of cataplectic attacks, but all we could elicit was that when he laughed heartily he "seemed as if he was doubling up." He did not actually "double up" but experienced the sensation of doing so. I have never heard of a syringomyelic process extending as high as the mid-brain or hypothalamus, but it does not seem impossible. Also, it is interesting to speculate how far a spastic condition below the level of the cervical enlargement of the spinal cord would modify the hypotonia of a cataplectic attack. There appears to be a very ill-defined dividing line between the somnolent attacks of narcolepsy and catalepsy. I have one case which I have watched for six years—in a man now aged 34—who has suffered from periodical attacks of prolonged sleep since 1919. The only physical signs present are slight ptosis and unilateral facial weakness; a radiogram of the skull shows an area of calcification occupying the approximate site of the pineal gland.

Sir James Purves-Stewart.

Dr. Wilson has pointed out that both in cataplexy and in narcolepsy, one of the most important and constant phenomena is muscular atonia. It has been recognized by artists for centuries that muscular atonia is one of the essential features in normal sleep. If we study pictures of sleeping children, or the sculptures of sleeping individuals through all the ages we find that all are characterized by muscular atonia. This atonia, whatever be its explanation, amply accounts for the loss of knee-jerks observed in cataplexy.

Dr. Wilson has referred to hypersomnia occurring in lesions in and around the third ventricle. The commonest pathological lesions are tumours somewhere around the third ventricle, most commonly below it, but sometimes at the sides or back, in the region of the corpora quadrigemina or the pineal body. In this connexion I may mention that if we inflate the third ventricle with air from above by ventricular puncture, the patient's outstanding symptom afterwards is that of happy, peaceful

sleep. Whereas if we inflate the ventricles from below, at the same time filling the subarachnoid space, very often there is a considerable meningeal reaction with headache, etc. I was able to demonstrate this last week in a man with a suprapituitary tumour, which had blocked off the ventricles completely from the cisterna magna and spinal theca. On replacing the ventricular fluid with air by ventricular puncture he fell sound asleep and slept for two days. Incidentally he was at once relieved from his headache.

Finally, there is one important point to which Dr. Kinnier Wilson did not refer. Why is it that we get these symptoms of sleep and narcolepsy paroxysmally instead of continuously? The paroxysmal character of the phenomena has not yet been explained. No stationary or static lesion in the floor of the third ventricle, in the mid-brain, or even in the higher centres, can adequately explain the paroxysmal character of the syndrome. There must be some superadded intermittent factor and this is a crucial point on which we desire information.

Dr. Wilfred Harris.

I have no personal experience of narcolepsy. "Somnosis" is a bad word, being a hybrid, half Latin and half Greek, that is why it has died. I do not quite follow what Sir James Purves-Stewart said about stationary lesions not accounting for paroxysmal attacks. There is nothing technically stationary in the human body. But in the ordinary sense we must agree that it is possible to produce paroxysmal phenomena from such lesions—I instance only paroxysmal trigeminal neuralgia. The phenomena occur day after day, year after year for perhaps fifty years, without any alteration whatever in the lesion.

Dr. T. Grainger-Stewart.

During the past two years I have seen three cases of narcolepsy. The case which is of special interest in relation to Dr. Wilson's remarks upon narcolepsy, catalepsy and epilepsy may be described as follows:—

The patient, a girl of 15, had suffered from epilepsy for several years. The fits were of the major type, with generalized convulsions, occasional biting of the tongue and passing of water, and they tended to be serial. They were controlled by luminal, bromide and belladonna.

Subsequently a different type of attack developed, which at first I thought was of the nature of petit mal. These attacks, however, had the following peculiarities:—

(1) They only occurred at meal-time when the child first took up her spoon or fork.

(2) There was no loss of consciousness; the patient always knew when she had had an attack, and said she would do anything to stop them, but she "just couldn't."

I specially observed her during an attack. As she raised the spoon to her mouth her eyes turned upwards and she suddenly collapsed, falling forward on the table and dropping the spoon. She recovered instantly and finished her meal without further difficulty.

During last summer these attacks practically disappeared and the patient seemed to be steadily improving. In the late autumn, however, I was asked to see her on account of "sleeping attacks," which occurred at irregular intervals, and which the parents thought might be due to encephalitis lethargica. These attacks lasted for

half an hour, or for two to four hours. Their onset was abrupt, but the child was not sleepy, for she was not unconscious and understood when spoken to. She lay in bed unable to move, with her eyes closed and her mouth open.

If the attack came on while she was eating the food remained in her mouth and saliva dribbled from the corners. If her eyelids were raised she could move her eyes, but she had practically no power in her legs or body. The limbs were atonic and flaccid, and all the deep reflexes were absent. The abdominal reflexes were sometimes present, but the plantar reflexes were abolished. Respiration was regular, but the respiratory movements were minimal. The rate did not increase. The temperature was normal or subnormal, but the pulse-rate always rose from 100 to 140 or over.

Her mother said the attacks passed off quickly, and though I was never present during the transition period, I have examined the patient half an hour after she has recovered from an attack, and found her bright and intelligent, with good power, and with all her reflexes present and normal. Her condition during an attack resembled that of a patient whom I saw at the National Hospital, Queen's Square, and who suffered from family periodic paralysis, although in that case the patient seemed to be mentally more alert.

Dr. P. C. P. Cloake.

I will give a patient's own description of a narcoleptic attack. This patient, a girl of twenty-two, suffered from primary narcolepsy.

"When I first noticed how sleepy I was feeling I was between the ages of 19 and 20. Even as I walked along I would fall asleep, for several times I did not even know how I had managed to get from one end of the workroom to the other, but would suddenly wake with a start and find I must have been asleep. For at least six months, every time I fell asleep and happened to be in a sitting position my head would fall right back as far as it was possible, then flop right forward for at least a dozen times, and I was quite unable to stop it. As time went on, I suddenly found that as soon as I lay down in bed I could not move or speak, and if I tried I could only make a muffled sound. But if anyone happened to come into the room and speak I could hear what they said quite well, and if they passed in front of my eyes I could see them, although my eyelids felt almost shut. I would have as many as six or seven straight off, but each one could have only lasted a minute or so. This last eighteen months these turns have got stronger, and it is more of an effort to pull myself round, as I have to make two or three attempts before I can do so, and then when I get up I usually jerk forward once or twice before I can steady myself. But for feeling a little out of breath I am quite all right.

"I fall asleep at all times, even standing up at work. I am so bad that I have to go and talk to someone or go out in the air before it passes off, and then, more often than not, the next half hour I am just as bad. This will keep on perhaps all day, and then next day I only feel like it once or twice. After the sleepy feeling passes off my eyes feel very sore. When I laugh, my legs give way and I have to hold on to anything or anyone near or I should fall. Also my eyes seem to reel about, for a minute or so all seems a blank, then everything seems muddled up and out of place and I have to shut my eyes before I can see plainly, and most times, instead of seeing one I see two. This also happens if anyone takes me by surprise and I turn round quickly. I have to wait a minute or so before I can speak."

This case illustrates the cataleptic, narcoleptic and cataplectic features of the disorder. I have heard sufferers from chronic encephalitis lethargica give identical descriptions of their cataleptic state.

I would emphasize the *associations* of the cataplectic attacks described above, because they show the essential similarity of these and the sleep attacks. Her eyes

"seem to reel about"; there is mental blankness followed by confusion and disorientation so that things "seem muddled and out of place"; then diplopia before clear vision. These features seem to indicate that a state of threatened sleep supervenes between the cataplectic attack and clear consciousness. On the other hand, Dr. Wilson's graphic description of the state of the body during cataplexy shows that it is similar to the somatic state in profound sleep, at least, in so far as universal atonia and extensor plantar reflexes are common to both.

Pavlov identifies sleep with a state of generalized internal inhibition of the cortex and some of the lower parts of the brain. One does not know, of course, what the neurological process of inhibition is. It might be merely a concealing or shunting of activity or an abolition of it.

Now there is no sharp line of demarcation between sleeping and waking. I think we can identify a complete series of mental states from highest mental activity to sleep itself.

At the top we have constructive thought which finds new solutions for new problems, next a level of habitual thought, a retraversing of familiar paths, serviceable for the regularly recurring problems of everyday life. The level below this is reminiscent thought, and next, reverie, in which the chain of associations is determined almost entirely by their intrinsic strength and not by any ultimate object of thinking which must be attained.

The associations become more free, logical connexions less apparent, attention less intense, and the mental content vaguer, as we pass down this series into sleep. In the process of falling asleep the mind follows this descending series and the process is thus revealed as the correlate of a gradual diminution of that neural energy which must underlie psychic activity in waking life; the general internal inhibition identical with sleep must bring about such diminution. Thus inhibition would appear not so much a process masking or shunting neural activity as one in which it is reduced or abolished.

Another point is whether such idiopathic narcolepsy is a disease. If cataplectic phenomena are not met with in symptomatic narcolepsy, is it not right to describe this association of cataplexy and sleep seizures as a disease? All diseases of unknown ætiology and pathology are syndromes of symptoms sufficiently associated and distinctive to make it probable that a definite, if unknown, pathologic process exists to particularize them.

Dr. Henry Cohen (Liverpool).

In discussing a comparatively rare phenomenon, valuable data must ensue from the pooling of experiences. I have notes in eight cases of narcolepsy, five "idiopathic" and three post-encephalitic. I shall not reiterate the details mentioned by previous speakers but shall confine my remarks to cases showing the close ætiological relationship between the narcolepsies and the epilepsies, a relationship denied by Adie in his recent thesis. Mrs. E. S., aged 56, whose brother died at 28 years, after typical epileptiform attacks during two years, has for the past six years suffered from typical narcolepsy, including both sleep and cataplectic attacks. The cataplectic attacks are precipitated by any sudden emotional stress or start, e.g., a loud, unexpected noise. Gordon Holmes[1] has recorded a similar case in which the cataplectic attacks were precipitated by noise alone, and in which typical epilepsy has since developed. Gowers and others have noted that the exciting cause of a true epileptiform attack may be a noise (the Lärm-epilepsie of the Germans). Recently

1 *Brain*, 1927, 1, 720.

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I have observed a boy, aged 13 years, who suffered from typical spastic diplegia from birth; at 5 years, for 12 months, he had typical epileptiform attacks with loss of consciousness; these ceased for two years; then he commenced to have attacks, two or three times daily, precipitated by a loud, unexpected noise, e.g., banging of a door, in which he fell, had myoclonic movements of face, jaw, arms and legs, a few seconds later, lasting 1 to 1½ minutes, during which he retained consciousness, could hear everything said to him, but sometimes could not see. He has no sleep attacks. I have seen this boy in an attack which closely resembles the experimental myoclonus described by Muskens. This case is, I take it, intermediate between the narcolepsy of my first patient and Lärm-epilepsie, and shows the link between these two conditions. Similarly the transition from the cataplectic attacks in narcolepsy precipitated by laughter, the Lachschlag of Oppenheim, and true epileptiform attacks precipitated by sudden emotional disturbance indicates that the mechanism producing the narcolepsies and the epilepsies is fundamentally similar.

Dr. Gordon Holmes.

I have seen two cases of true narcolepsy, one of which Dr. Wilson examined with me a few weeks ago. That was in a boy whose condition was in every way characteristic. It brought out a fact of which I was not aware before; this boy had been for years a subject of nose-bleeding and as the narcolepsy began, the nose-bleeding ceased. Dr. Wilson says that this has frequently been observed, but what the relationship is I do not know. The second case was in a highly educated schoolmistress, aged 24. She was subject to attacks of both kinds. Curiously enough, in her case the cataplexy occurred more readily on annoyance than on laughter. She used occasionally to collapse in class if annoyed with a pupil.

The most interesting case I have seen in relation to this subject is one that we should probably speak of as symptomatic- rather than idiopathic-narcolepsy. The patient was a middle-aged clergyman, who for some years had been greatly increasing in weight, and for some time before I saw him had suffered from headaches. Irresistible attacks of sleep set in, and he described as "agony" his attempts to write letters, to keep awake during religious services and other duties. Here was undoubtedly a local disease, because in the upper right quadrants of both visual fields was a small scotoma. I believe this man was the subject of a tumour in the region of the hypothalamus, involving the optic tracts rather than the optic radiations. He was also subject to attacks of falling. These first occurred some months before I saw him. He was watching a football match and the boys of his school scored; he fell down with excitement. These attacks were not, however, frequent in his case.

Here we have practically a full picture associated with a lesion in the brain. Even more interesting is the fact that a chance measure relieved both symptoms. I suggested thyroid in the absence of any known therapy, and after four grains daily had been taken for a short time the attacks became much less frequent and eventually ceased. It is, however, possible that this favourable result may have been independent of the therapeutic measures employed, as possibly the neoplasm has now reached such

an extent that it has destroyed other centres, or is interfering to a greater degree with the functioning parts.

The fourth case was in a boy whom I showed here in July, 1927. His only symptom then was that on a sudden noise his limbs relaxed and he fell, a mass of toneless muscle. We had an opportunity of examining him on several occasions, and Dr. Critchley ascertained that in these attacks his reflexes disappeared. He was then aged about 15, and from the age of two had been subject to these attacks. On the other hand, he had never been subject to sleeping attacks. A few days before I showed him he had had one epileptic seizure in the wards of the National Hospital. Since then, however, he has had a large number of epileptic seizures. This case is possibly allied to narcolepsy, although sleep has never been a prominent feature in it

Dr. R. D. Gillespie.

Dr. Kinnier Wilson had pointed out that cataplectic attacks were frequently precipitated by such minor emotional disturbances as were associated for example, with smiling. It was curious that in such patients, presumably powerful emotional situations tended not to be followed by a cataplectic attack. Thus, a patient whose case had been recorded by the speaker in the *Journal of Neurology and Psychopathology* did not have an attack while rescuing a man from serious injury on the railway; but he had a mild attack when he thought about it afterwards. Similarly, when suddenly made very angry on one occasion, no attack followed, although he had been persistently subject to cataplectic attacks on the slightest emotional provocation. Not much had been said of therapy in narcolepsy, because there was so little to say. The case mentioned, not being at first recognized for what it was, was treated for a time by psychotherapy and the patient had remained free from attacks for two months, only to relapse completely afterwards. Bromide and luminal having produced no impression hydrobromide was tried as a desperate remedy; the cataplectic attacks had greatly diminished in frequency, but the tendency to drowsiness had increased. Another patient felt slightly less inclined to be drowsy on caffeine. In one of the cataplectic attacks of the first patient mentioned there was loss of knee-jerks, and the right plantar response was flexor, the left being absent. When he was drowsy his left eyelid was slightly ptosed and the corresponding pupil sluggish to light.

It was difficult to regard any of the attacks as paroxysmal, as Sir James Purves-Stewart had suggested. One patient felt the narcoleptic attack coming on all forenoon, the attack itself being a climax which she could no longer resist and which was followed by a feeling of relief. Attacks of cataplexy, on the other hand, were occasional in origin. The apparent relation of attacks to vasomotor conditions was interesting; the first patient mentioned was more liable to cataplectic attacks after a hot bath, while he could at times avert an attack by dashing cold water on his face. During narcoleptic attacks automatic movements such as writing were sometimes performed, a fact of interest in view of the possible transitions to epilepsy.

Dr. KINNIER WILSON (in reply).

Both cataplectic and narcoleptic attacks have been described in post-encephalitic cases. That is therefore a strong argument against the view that narcolepsy should be regarded as a "morbus sui generis." Every gradation can be found from narcolepsy without cataplexy, to cataplexy without narcolepsy. Narcolepsy is linked definitely to the epilepsies on the one side and the catalepsies on the other.

Section for the Study of Disease in Children.

President—Mr. GEORGE E. WAUGH, F.R.C.S.

[*March* 23, 1928.]

CASES.

Atrophy of the Parotid Salivary Gland.

By JAMES MAXWELL, M.D.

A. E., AGED 8½ years, male.

The boy was first brought to hospital in October, 1921. He then had an enlarged and tender gland below the left mastoid process, near which was a sinus at the site of an incision made into an abscess five weeks previously. A diagnosis of tuberculous glands with a tuberculous sinus was made, and general treatment was carried out for a few months. At the end of January, 1922, the local condition was definitely worse, and X-ray treatment was advised. This was carried out only from January to April, 1922, after which the discharge from the sinuses ceased and healing occurred. The patient was then lost sight of for three and a half years, but in September, 1925, he again came under observation, being considerably under-sized and under-weight. Thyroid extract was given on the assumption that the X-ray treatment might have inhibited his normal growth through the thyroid gland, and improvement resulted.

In May, 1926, he is said to have had mumps.

He reappeared in November, 1927, when facial asymmetry was noticed.

Present Condition.—Under-sized and thin. Weight, 49¼ lb. Below the angle of the left mandible are the scars of the healed sinuses; a few small discrete glands are now palpable in this region. The lower and posterior part of the parotid salivary gland is completely atrophic, with a resultant hollowing of the cheek on the affected side. No flow of saliva has been observed from the left Stenson's duct, but the mouth is not abnormally dry.

I think it will be agreed that this is atrophy of the parotid salivary gland. It has been suggested that the masseter is wasted also, but the jaw seems to move freely in all directions.

There are two possible causative factors. The first is the X-ray treatment on that side of the neck; the second the mumps, which he is said to have had in 1927. The mother said that wasting was noticed before the mumps appeared, and that when he had mumps the swelling was confined to the opposite side of the face. Dr. Finzi agreed that the atrophy was probably due to the X-ray therapy. I do not think there is any necessity for treatment.

Discussion.—Mr. G. E. WAUGH (President) asked whether it was thought that the X-rays destroyed the lower part of the gland, or that the destruction was caused by infective processes. He had tried to destroy the parotid gland in a child who had a salivary fistula from a birth injury, but X-rays, given to the maximum within the safety margin, had not influenced the function of the gland at all.

Dr. MAXWELL (in reply) said he did not think there was any evidence of inflammation in the parotid region, therefore the atrophy was much more likely to have been due to the irradiation. Had it been caused by infection he thought there would have been a swelling or some evidence of local induration.

Renal Infantilism.

By E. A. COCKAYNE, D.M.

A. B., MALE, aged 5 years. Weighed 8 lb. at birth. Breast-fed for three months. Growth was always slow, and his weight at 2 years was 17 lb., and at 5 years 26 lb. 15 oz. There is an elder child aged 6 years and a younger sister aged 1 year 8 months; both are normal, and the latter weighs 26 lb. 8 oz. His mother states that he has been very thirsty for the last three years at least, and drinks up to six pints a day. Under observation in hospital he did not appear to be unusually thirsty unless excited, but all specimens of urine, including those passed in the early morning, were of very low specific gravity, 1010 or less. Pituitrin had no effect either on the amount of urine or on the specific gravity, as the following table shows:—

	Daily water intake in oz.		Amount of urine		Specific gravity
No pituitrin	30	...	22	...	1010
,,	60	...	24	...	1008
,,	60	...	30	...	—
,,	76	...	60	...	1001
,,	72	...	60	...	1005
Pituitrin 0·5 c.c.	99	...	90	...	1005
,,	62	...	46	...	1005
,,	96	...	42	...	1006
,,	58	...	32	...	1007
,,	46	...	21	...	1005
Pituitrin 1 c.c.	50	...	42	...	1005
No pituitrin	47	...	41	...	1005
,,	65	...	32	...	1005
,,	66	...	30	...	1005

Urea-concentration Test.—Before administration of 8 grm. of urea, 1·45 per cent. urea; first hour, 1·65 per cent. urea, 67 c.c. urine; second hour, 1·85 per cent. urea, 79 c.c. urine: third hour, 1·36 per cent. urea, 87 c.c. urine. Blood-urea, 42 mgm. per cent. A trace of albumin was found in one specimen of urine, but none of the other specimens examined had any; no casts have been found, and only a few white blood-cells. Mentally he is rather precocious, and physically very active. Except for his small size, and the fact that his heart's apex beat is just outside the nipple line, no abnormal physical signs have been discovered. Discs, normal. Wassermann reaction of blood, negative. X-ray shows a normal pituitary fossa with no evidence of tumour, and long bones normal.

The excessive thirst was probably largely a matter of habit. I do not think the condition is diabetes insipidus; he does not pass a huge quantity of water, and

pituitrin had no effect. The most probable diagnosis is that of renal infantilism. He is small for his age, about the same size as his sister aged 2, and his urine has always a low specific gravity. There has only once been albumin, and there are no

FIG. 1.

casts, but there is rather a high urea-content in the blood, and there is only a poor power of urea-concentration. I think that later he will develop definite signs of renal disease. I show his photograph with that of an average boy of the same age for comparison.

Enlarged Mediastinal Glands.

By DONALD PATERSON, M.B. (shown by Dr. R. COVE-SMITH).

BOY, aged 6 years. Has attended hospital intermittently since age of 20 months, when he had a small suppurating gland in the right cervical region following diphtheria. This gland was excised.

52 Paterson: *Enlarged Mediastinal Glands*

At the age of 3 years he had a swelling on the left side of the neck and was reported to be "out of sorts." No abnormal physical signs were found in the heart or lungs, but he was said to have a night cough and to sleep restlessly.

At the age of 4½ years he was brought up because of a cough. Some "bronchitis" was said to be present and he was noted to breathe in a very shallow manner.

At the age of 5½ years (March, 1927) there was still complaint of cough and he only gained one pound in three months (46½). He was reported as looking "below par." In August, 1927, he had whooping-cough and was not seen again until February, 1928, when the following physical signs were present in the chest: Catarrhal râles all over both lungs, but more marked at the right base where

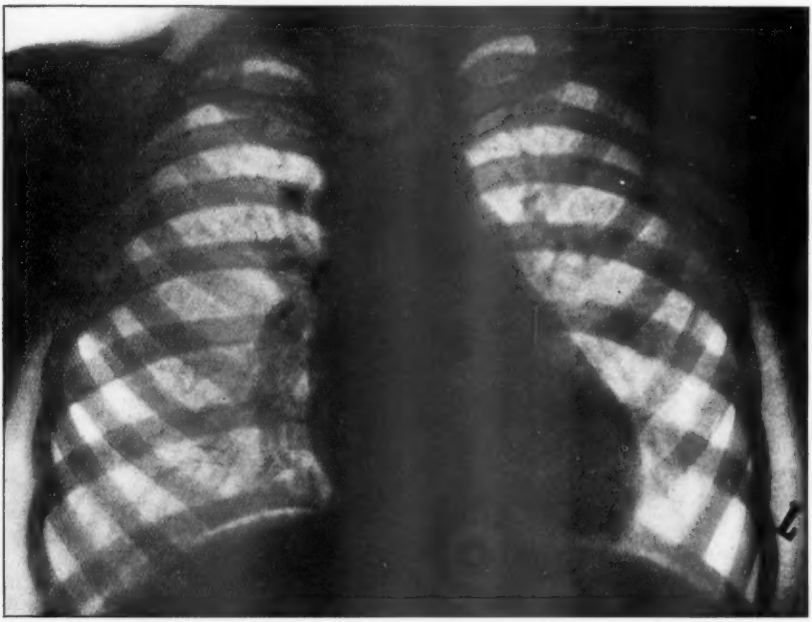

FIG. 2.

expansion was poor. The percussion note was impaired to the right of the vertebral column while prolonged expiration and clicking râles are present in both axillæ, though more marked on the right than on the left. Intracutaneous tuberculin test (Mantoux) strongly positive. X-ray: Mediastinal glands +: many calcified in both lung roots.

The condition is rather common, but is not diagnosed so frequently as it might be. The attack of whooping-cough in August, 1927, apparently lit up latent disease in the hilar glands. The point at issue is whether one should treat this child merely as a catarrhal case, or as a possibly incipient tuberculous case; in view of the history my own leaning is towards the latter.

Discussion.—Dr. DONALD PATERSON thought that Dr. Cove-Smith was serving a useful purpose by bringing this case, as such cases were brought for discussion too seldom. He thought that the glands revealed by the X-rays were really tuberculous, that it was this type of case which gave rise to miliary tubercle or spread to the lungs, and in which the child developed the adult type of tuberculosis. The Mantoux test, which had been employed far too little in this country, especially in London, was, he considered, a valuable aid to diagnosis in such a case as this.

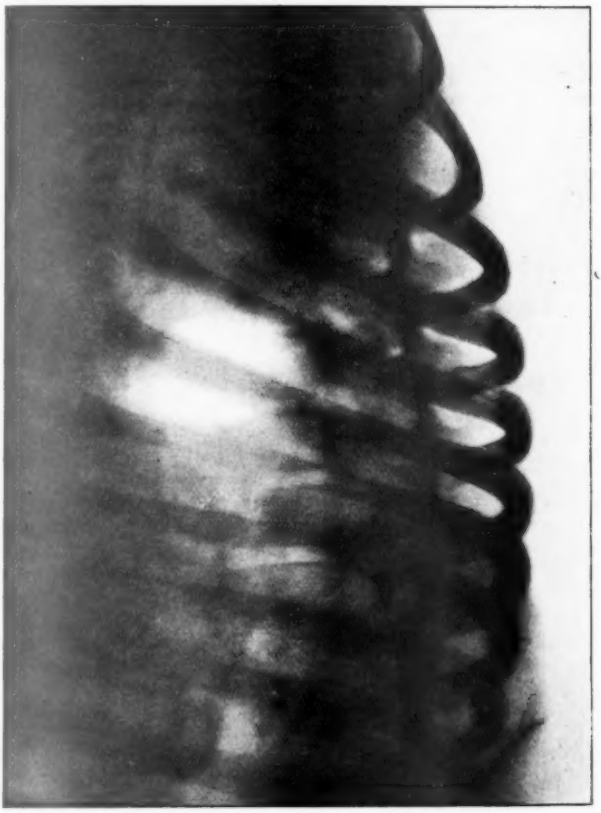

FIG. 3.

Dr. A. A. MONCRIEFF said that so long as the tuberculosis was limited to the glands, the treatment was the same, whether the enlargement was simple or tuberculous: it was only the prognosis which differed.

Dr. J. MAXWELL asked whether the Mantoux test showed a tuberculous infection which might or might not be active, or whether it showed a definitely active tuberculous infection. Also, did it apply only to children, or was it of value in adults as well?

54. Worster-Drought and Danvers-Atkinson: *Juvenile Tabes Dorsalis*

Dr. COVE-SMITH (in reply) said that he had employed the Mantoux test only on children. His own Mantoux reaction was not strongly positive.

He agreed that the treatment was the same, no matter what the exact form of the disease. He had brought the case because so many rarities were seen at the Meetings that typical and more ordinary ones were apt to be overlooked.

Juvenile Tabes Dorsalis.

By C. WORSTER-DROUGHT, M.D., and F. C. DANVERS-ATKINSON, M.B.

J. F., MALE, aged 13 years, was admitted to hospital on February 28, 1928, complaining of failure of vision, especially in the right eye, for two years.

Present Illness.—Two years ago it was noticed at school that he could not see the board and his eyesight was then investigated. He has been under treatment since then and the sight of the right eye has improved a little as a result.

Previous Health.—No illnesses of importance.

Family History.—His mother died six and a half years ago. For two and a half years before her death she was mentally afflicted and was confined to a mental hospital (? G.P.I.). She had four children including the patient. The eldest is 27 years of age and in good health; the second was born dead twenty-five years ago; the third, born twenty-two years ago, died of rheumatic heart disease at the age of twelve. The father admits infection with syphilis twenty-two years ago. His blood Wassermann reaction is now strongly positive.

Condition on Examination.—The patient is a small, rather undersized boy. The bridge of his nose is somewhat sunken: face drawn and tired looking, and expression rather older than usual in a boy of his age. The four upper incisors are narrow and peg-shaped; the first and second incisors on the right side are notched on the cutting surface. Both upper first molars are deformed and approximate to the shape of a truncated cone. There is bossing of the frontal and occipital bones.

Central Nervous System.—Right pupil is moderately dilated, round, situated nearer to nasal than to temporal side, and smaller than left. It fails to react to light and consensual stimulation, but reacts sluggishly to accommodation. Left pupil dilated, round and eccentric; fails to react to light or to consensual stimulation, but reacts sluggishly to accommodation. Vision is much impaired in both eyes; right eye, failure to see fingers but perception of light still present; with left eye he can read large print at 10 in. The visual field of the left eye apparently normal. Both optic discs show optic atrophy, more advanced on right side than on left. The discs are clearly defined, pale white in colour and surrounded by black pigment; the physiological cup is obliterated, and the vessels extremely small. In the retina the vessels appear as faint shadows, and lymphatic sheaths are obvious on both sides of them. The retina is peppered with small dark areas, and occasional masses of black pigment are seen: these changes being more obvious on the right side. Bilateral ptosis, more marked on left side, and on both sides fully corrected on upward deviation of eyes by compensatory action of the frontalis. Speech is normal. Hearing is apparently normal, but on both sides bone conduction persists longer than air conduction. Corneal reflex is normal on right side; diminished on left. Voluntary and associated movements of facial muscles are full and equal on the two sides: but emotional movements are somewhat deficient on left side. Other cranial nerves normal. Sensation for cotton-wool normal. Sensation for pain impaired on ulnar aspect of upper half of right forearm: and on ulnar aspect of left arm and forearm. Deep pressure sensation normal on right side, and impaired over lower end of left radius and ulna, also in left tendo Achillis. Sense of posture impaired in

fingers and toes on both sides. Localization impaired in the analgesic areas. Appreciation of vibration and recognition of shape normal. In both upper limbs there is an appreciable diminution in muscle tone, but no wasting; motor power normal and equal on the two sides. Supinator jerks absent; biceps and triceps jerks present and equal. In lower limbs, motor power and muscle tone normal, and no wasting: knee-jerks present and equal; ankle-jerks absent, and plantar reflexes flexor with brisk withdrawal movement. All abdominal reflexes present and equal on the two sides. Gait: He walks on wider base than normal. Coördination: Finger-nose test normal with the eyes open, but slightly impaired with eyes closed; on standing, slight swaying appears with eyes closed. Sphincters normal.

Cerebro-spinal Fluid.—Cells, 28 per c.mm.; practically all small lymphocytes, with a very occasional small endothelial cell and a very occasional polymorph; total protein, 0·04 per cent.; globulin, a trace in excess; Wassermann reaction strongly positive: Lange's colloidal gold reaction, 1222210000.

Other Systems.—Heart: Apex beat in fourth intercostal space 3 in. from middle line with pulsation in second left intercostal space; in mitral area, the beginning of first sound is rough, with soft systolic murmur and a faint late diastolic murmur; in aortic area there is a soft systolic murmur. Peripheral blood-vessels normal; pulse, 84, regular; blood-pressure, 135 systolic and 85 diastolic. Respiratory and vasomotor systems show nothing abnormal. Wassermann reaction in blood strongly positive.

Commentary.—Tabes dorsalis is a rare condition in childhood, and is considerably less frequent than juvenile general paralysis. The first recorded case appears to have been reported by Henoch in 1875. The difference between juvenile tabes dorsalis and Friedreich's ataxia, however, was not always apparent to the earlier observers until Hildebrandt called attention to the error and drew the distinction between the two conditions. The recorded cases are not numerous; the late Mr. Sydney Stephenson showed a patient, aged 9 years, before this Section on November 26, 1915,[1] and one of us (C.W.-D.) showed a girl, aged 10 years, in 1924.[2] This latter patient is still doing well; she has grown into a tall girl of healthy appearance, complaining of no symptoms of her diseases, although she continues to show double Argyll Robertson pupil with absent deep reflexes, and a positive Wassermann reaction in the blood and cerebro-spinal fluid. She has been treated with courses of sulfarsenol and colloidal mercury injections.

The present case is below the average age incidence of the recorded cases (15 years) and the disease seems to have made its first manifestations two years ago—when the patient was aged 11. The youngest recorded case is that described by Mingazzini and Bascheri-Salvadori, in which the onset occurred at 3 years of age. Features of interest in the case shown are: (1) The family history—the mother apparently dying from G.P.I., and the father having a positive Wassermann reaction in the blood. We have since had the opportunity of examining the father; he shows no physical signs of nervous disease, and no evidence of syphilis beyond the positive Wassermann reaction. (2) The changes in the fundus oculi. (3) The retention of the knee-jerks with absent ankle-jerks. Apart from the risk of intercurrent infection, such as pneumonia, the prognosis of juvenile tabes under treatment does not appear to be bad.

Discussion.—Dr. F. PARKES WEBER referred to cases of children in whom, besides evidence of congenital syphilis, there was no sign of disease to be discovered, excepting more or less optic nerve atrophy. He asked Dr. Worster-Drought whether these children should be classified as "candidates" for juvenile tabes dorsalis—that is almost the same as to say "incomplete forms."

[1] *Proceedings*, 1915-16, ix (Sect. Study of Dis. in Child.), 13.
[2] *Proceedings*, 1924, xvii, *ibid.*, 18.

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Dr. W. S. C. COPEMAN asked whether it was possible for this type of condition to arise as a sequel of third-generation syphilis. A case came before the Courts in which there was a great difference of opinion among experts.

If the possibility of third-generation infection was allowed, what was likely to be its clinical form in the majority of cases?

Dr. WORSTER-DROUGHT (in reply) said that he would class the patient described by Dr. Parkes Weber as an early case of tabes, or, as Dr. Parkes Weber himself put it, he would regard him as a candidate for tabes later. The boy might possibly develop general paralysis instead of tabes, as the actual disease-process was the same, the difference depending upon whether the brunt of infection fell on the cord or on the brain; it was difficult in some cases to draw a hard-and-fast line between the two conditions. When optic atrophy was pronounced it was more likely that the case would be one of tabes rather than of G.P.I.

In reply to Dr. Copeman, he had never seen a third-generation case of tabes dorsalis, but he had seen one of congenital syphilis, so it might be possible for tabes to result in a third-generation case. The case he had in mind at the moment was that of a family in which the grandfather had tabes, the son had disseminated choroiditis, optic atrophy and Hutchinsonian teeth, with a strongly positive Wassermann reaction, while the grandchild, aged 2, had a depressed nasal bridge, frontal bossing and other signs of congenital syphilis, with a positive Wassermann reaction, but, so far, no definite evidence of nervous involvement.

Abscess in Frontal Lobe; Post-mortem Findings in Case shown at a Previous Meeting of the Section.

By D. W. WINNICOTT, M.R.C.P., and ELIZABETH O'FLYNN, M.R.C.P.

G. C., AGED 6 years at death, had been under observation at The Queen's Hospital for Children since February, 1925. Besides the odd complaints from which she suffered it was always felt that some more important disease might be present. As her mother has active phthisis, T. B. was suspected, and as the child had severe headaches and seemed not to progress normally in mental development intracranial tuberculoma was thought of, but not localized. There was no other sign or symptom of intracranial disease, except a change in fat distribution and a certain amount of so-called sexual precocity, which became evident about January, 1926. A search was made for further evidence of pituitary disease, but no clue was discovered. The child always did well in hospital and at the convalescent home. The chief minor complaint was of an offensive vaginal discharge. She used to wake at night screaming with head pains.

In November, 1926, she had whooping-cough very badly, but although she became very weak she did not waste at all, but became even fatter and showed the puberty type of distribution of fat (see photograph).

In January, 1928, acute symptoms pointed directly to intracranial disease, and T. B. meningitis was expected. Lumbar puncture did not confirm this diagnosis, but the patient died before further investigations could be made, January 31, 1928. There was no vomiting.

Five days before death there was slight papillœdema on the left side. Otherwise no C.N.S. alteration was discovered, except absent left upper abdominal reflex, noted by the H.P. a few hours before the child died.

Pathological Findings: January 25, 1928.—Leucocyte count = 30,100, of which 80 per cent. were polymorphs.

C.S.F., January 26, 1928.—A blood-stained C.S.F. Chloride = 0·73. Urea = 45 mgm. per cent.

C.S.F., January 30, 1928.—Clear, colourless fluid. Fibrinous flakes present. Globulin, Nonne-Apelt weakly positive.

Cells 120 per c.mm.; 70 per cent. polymorphs; 30 per cent. lymphs; chloride 0·68.

Culture: streptococci reported after death of patient.

FIG. 1.—Showing abscess in right frontal lobe.

Post-mortem Report.—*Brain*: A large abscess is present in the right frontal lobe involving the whole anterior half of the right hemisphere. There is considerable flattening of the convolutions. The cavity abscess is about 3 in. deep

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by 2 in. wide and extends from the vertex to the orbital surface of the frontal lobes, where it almost comes to the surface. The cavity contains a quantity of blood-stained pus. A streptococcus is present in direct films, and also in culture.

FIG. 2.—Showing the distribution of body fat, and the false appearance of good health at a time (summer, 1927) when the child was constantly ill with fever and severe headaches.

Sinuses: ethmoid, sphenoid, mastoid, all appeared healthy, and no obvious primary focus found.

Kidneys: extremely congested.

Nothing else of importance, apart from enlarged thymus and lymphoid spleen.

The interest of this case lies in the following points:—

(1) The difficult clinical picture presented both in the long chronic stage and in the acute exacerbation which led to the death of the patient.

In the chronic stage the patient was shown to the Members of this Section, but as no help in diagnosis was offered the notes were not published.

(2) The change in distribution of fat, illustrated in the photograph, which gave a false impression of extreme health even when the child was very weak and ill.

Muscle Weakness, altered Gait and absent Deep Reflexes after Measles.

By D. W. WINNICOTT, M.R.C.P.

FEBRUARY 20, 1928. J. W., aged 6 years, was brought to the Queen's Hospital for Children because of weakness and inability to stand.

Past History.—Diphtheria at 20 months. Tonsils and adenoids removed.

Family History.—Only child of healthy parents. No miscarriages.

History.—Quite well till measles Christmas, 1927, for which he was in bed two weeks. When he got up he was found to be very weak, more so than could be accounted for by the general condition. The weakness grew worse, so that by February 20 (when first seen) he had to hold on to something in order to stand. Apart from this and getting thinner, he seemed to be well.

He notices there is something wrong, and says: "My top is too heavy for my legs." He has had no pains or paræsthesiæ.

Examination.—The limbs are wasted. The gait is that of weak muscle tone. The only abnormalities found are symmetrical weakness of the muscles of the limbs, with absent tendon reflexes, and slight foot-drop.

No sensory disturbance can be made out; there are no trophic skin lesions; sphincter control is normal. There is no muscle tenderness.

Electrical reactions: All muscles tested respond to faradism, with diminished response in the dorsiflexors of the feet.

March 12, 1928.—There is some improvement in gait and in strength of grip. Tendon reflexes absent.

It seems likely that the condition cannot be explained by simple weakness after illness. There must be a peripheral neuritis. The boy walks and climbs up himself on rising from lying on the floor like a case of muscular dystrophy. But against this diagnosis is the quite definite improvement which has taken place in the last two weeks.

Dr. C. C. WORSTER-DROUGHT said he thought the condition was one of polyneuritis, or more strictly polyneuronitis, since the motor fibres were mainly affected, the sensory fibres having been spared. Peripheral neuritis followed most of the acute specific fevers, and it was recognized that meningitis and myelitis were among the sequelæ of measles.[1] It was scarcely necessary to postulate diphtheria; he did not see why polyneuritis should not follow measles.

POSTSCRIPT.—By the middle of April, 1928, the gait had become nearly normal, and the boy no longer climbed up himself (as in a muscular dystrophy). Knee-jerks were still unobtainable.

[1] Most text-books place neuritis in the list of complications of measles, and W. Harris ("Neuritis and Neuralgia," 1926) mentions measles among the the causes of infective polyneuritis.—C. W.-D.

60 Pearson: *Myatonia Congenita with Delayed Closure of Fontanelle*

Case of Myatonia Congenita (Oppenheim) with Delayed Closure of the Anterior Fontanelle.

By W. J. PEARSON, D.S.O., M.C., M.D. (shown by Dr. R. C. LIGHTWOOD).

O. S., NOW aged 2 years and 4 months, attends as an out-patient at the Hospital for Sick Children, Great Ormond Street, London.

History.—She was born prematurely at eight months, the birth-weight being just under 4 lb. A breech-labour was difficult, and the mother recounts that the baby was born with her legs black with bruises. The breast failed after a few days and the baby was reared on Allen and Hanbury's food till the age of seven months when she was given cow's milk; mixed feeding commenced at eleven months. She sat up at ten months, and said, "Mum" and "Dad" at the same age, but has never walked. She is a clean baby, napkins having been given up altogether at fifteen months.

She is the first child of two, the younger, who is now 8 months old, is a healthy though small baby, and her muscles are said to be firm.

Physical Examination.—The muscles show a very considerable lack of tone. The mother is quite certain that this softness of the muscles has been present from birth. The knee-jerks are obtainable. All the organs are normal. The head is of the rachitic shape and the anterior fontanelle is wide. Skiagrams of the bones show no evidence of any rachitic change. There is no sweating of the head neither are there any signs pointing to rickets other than the shape of the head and the open fontanelle. The skin is fine and soft and appears to have a normal tone. The mother says that the baby bruises easily. The Wassermann reaction is negative.

Discussion.—Dr. H. F. TURNEY (in Dr. Lightwood's absence) said: Rickets has been suggested here on account of the late closure of the fontanelle. The condition has been present since birth, and has not been improved by several months' treatment on cod-liver oil and sunlight. Skiagrams show no departure from the normal. The chief reason for bringing the case is to obtain ideas as to prognosis. I have never seen an adult with this condition; the oldest subjects I have seen have been aged about 12, and have suffered from serious disability in walking.

Dr. WORSTER-DROUGHT said he agreed that this condition was myatonia congenita. The degree of hypotonia was too great. An unusual feature was that the knee-jerks were retained.

With regard to prognosis, he had followed up some cases during a number of years, and his view was that the outlook was not so favourable as the text-books led one to believe. He thought this child would walk eventually, but that the gait would be feeble and waddling in character. He had never seen a case of the kind recover completely.

Hypertrophic Pyloric Stenosis in Uniovular Twins.

By E. A. COCKAYNE, D.M., and W. P. H. SHELDON, M.D. (for Dr. R. HUTCHISON).

THE patient and his twin brother were born on January 17, 1928. The attending midwife reported that there was one placenta, one chorion and two amniotic sacs. Both parents are healthy, and there has been one previous pregnancy, resulting in the birth of a male child, who is now 12 months old and quite healthy. The patient began to vomit when a month old, and was admitted to the Hospital

for Sick Children a week later. The diagnosis of hypertrophic pyloric stenosis was made, and at operation hypertrophy of the pylorus was found. Rammstedt's incision was performed, and an uneventful recovery ensued.

In the other twin, symptoms commenced at the age of 3 weeks and a week later he was admitted to hospital. The diagnosis of hypertrophic pyloric stenosis was made, and operation on the day after admission confirmed the presence of the typical tumour at the pylorus. Rammstedt's incision was performed, but unfortunately the infant succumbed six days afterwards.

One twin was 1 lb. lighter than the other; the one operated upon first was the lighter twin, and it was this one who died. Presumably the midwife's statement that there was one placenta, one chorion and two amniotic sacs was evidence that the twins were uniovular.

Familial Cases of Hypertrophic Pyloric Stenosis.

By E. A. Cockayne, D.M., A. A. Moncrieff, M.D., and W. P. H. Sheldon, M.D.

No history of other cases in the family. Parents unrelated. First child, a girl, born in 1917, normal.

Second child, G. B., a boy, born 1918. Bottle-fed. Symptoms noticed at 3 weeks of age. Rammstedt's operation at 10 weeks by Mr. L. E. Barrington-Ward. Diagnosis confirmed at operation. Cured.

Third child, R. B., a boy, born 1920. Bottle-fed. Symptoms noticed on fifteenth day. Rammstedt's operation on nineteenth day by Mr. L. E. Barrington-Ward. Diagnosis confirmed. Cured.

Fourth child, L. B., a boy, born 1921. Breast-fed. Symptoms first noticed on twelfth day. Projectile vomiting. Admitted to hospital, where the tumour could not be felt and no peristalsis was seen. Treated medically and cured. Although the mother is convinced that this boy had the same disease as his elder brothers the diagnosis was not established.

Fifth child, a boy, born 1922. Normal.

These two families have been brought before the Section because they throw light on the ætiology of congenital pyloric stenosis. Strachauer states that the lesion has been found in a seven months' fœtus and in the stillborn, and this goes far to prove that it is a developmental error.[1] Caulfield has collected evidence of its familial incidence. He cites several families in which two brothers or sisters were affected, six in which three were affected, and one, Finkelstein's, in which four were affected, and gives references to three examples of the disease in twins.[2] The condition is probably inherited as a Mendelian recessive, but this cannot be proved unless, in addition to the familial incidence, congenital pyloric stenosis is found to occur more often in the families of parents related by blood than chance intermarriage would account for.

1 Strachauer, A. C., *Annals of Surgery*, 1927, 167.
2 Caulfield, E. J., *Amer. Journ. Dis. Child.*, 1926, xxxii, 706-708.

Discussion.—I am afraid I have misled the Section in stating that three boys in this family had pyloric stenosis. I had only seen the two eldest, and the mother told me that the third had also been operated on. She told me to-day that she thought the operation in his case was an internal one. A diagnosis of pyloric stenosis based merely on the symptom of projectile vomiting cannot be accepted.

A Case of "Pink Disease" Four and a Half Years after Recovery.

By Donald Paterson, M.D.

V. L., a girl, aged 5½ years. At the age of 1 year this child was admitted to the Hospital for Sick Children, Great Ormond Street, under Dr. Still, for typical "pink disease." She had then been "irritable" for some months and the "rash" had been present on the hands for a few weeks. She was in hospital for four months and was very ill with diarrhœa at times. Patches of analgesia were present and the knee-jerks disappeared for some weeks. Lumbar puncture was performed, but the fluid was normal: Von Pirquet's test negative. Rapid improvement eventually took place. In 1925 she was re-admitted under Dr. Hutchison at the age of 3½, for acute cerebellar ataxy from which she completely recovered. At the present time she is perfectly well, except for a recent deafness which is receiving attention. Her reflexes are normal and the skin shows no permanent changes.

This is one of the original cases of "pink disease" shown at this Section. It was a very typical case of polyneuritis, and it conformed to the classical description. The child had a curious illness, of which I have no explanation, namely, acute cerebellar ataxy, two years ago. I cannot think there is any connexion between that short illness and pink disease. To-day, i.e., four and a half years after the illness, the child seems normal and is shown to demonstrate that fact.

Discussion.—Dr. F. Parkes Weber asked what the symptoms were in 1925, when the child was supposed to be suffering from acute cerebellar ataxy. Might not the atactic symptoms have been due to a kind of polyneuritis?

Dr. Tallerman said that when this patient was ill with acute cerebellar ataxy he (the speaker) was house-physician to Dr. Hutchison, and the only symptoms then were marked incoördination (the left arm, so far as he recollected, being worse than the right), and inability to walk properly owing to ataxia. The child was in hospital about six weeks, and at the end of that time she had recovered.

Dr. Donald Paterson (in reply) said that in the cases—about twelve—of pink disease that he had seen, the children appeared to have completely recovered after the subsidence of the initial symptoms. He did not see this patient when she had the acute cerebellar ataxy, but those who had the care of her were fully alive to the possibility of what Dr. Parkes Weber had mentioned. He did not think it was likely to have been a polyneuritic ataxy.

A Case of Intra-uterine Fracture of the Tibia and Fibula.

By B. Whitchurch Howell, F.R.C.S.

History.—M. T., aged five months, sustained an intra-uterine fracture of the lower third of the right tibia and fibula.

Present Condition: March, 1928.—Malunion of the tibia and fibula, with abduction, and backward displacement of the lower ends, resulting in marked talipes calcaneo-valgus, and shortening.

Suggested Line of Treatment.—(1) Double-wedged osteotomy of tibia with base inwards and backwards. (2) Subperiosteal fracture of fibula.

There is a definite shortening of the leg on the affected side, with perhaps an increase in the length of the femur on that side.

Is there any correlation between congenital clubfoot and intra-uterine pressure due to diminution of the amniotic fluid? The child has a definite talipes calcaneo-

FIG. 1.

valgus, and the common cause of that, in its pure condition is a small fœtus in a small uterus, with little amniotic fluid, in a small woman. I am wondering whether this case is one of true pressure-fracture from lack of amniotic fluid, leading to two deformities: talipes calcaneo-valgus and the fracture *in utero*. There is no history of injury to the mother before the birth of the child. Instruments were not used at the birth.

64 Whitchurch Howell : *Intra-uterine Fracture of Tibia and Fibula*

As to treatment, I have suggested a double-wedge osteotomy of the tibia with greenstick fracture of the fibula, maintaining the correction in plaster of Paris. I do not think it necessary to put in an intramedullary peg.

FIG. 2.

Discussion.—Dr. W. S. C. COPEMAN asked whether there was definite evidence that this injury had been intra-uterine ; he thought it might have been produced after birth.

Mr. G. E. WAUGH (President) said he agreed with the proposed line of treatment, as the deformity would be permanent unless interference was carried out now. He would suggest the insertion of a medullary graft at the same time. The operation was very difficult.

Mr. WHITCHURCH HOWELL (in reply) said that the case was first seen in his (the speaker's) absence, and he took it for granted that the history supplied to him was accurate. He would leave the title as it originally stood.

Swelling in the Region of the Tendo Achillis.

By B. WHITCHURCH HOWELL, F.R.C.S.

Complaint.—A. S., AGED 13 years, has an elastic swelling in the region of the right tendo Achillis.

Onset.—Congenital.

Past Treatment.—Partial removal at a hospital in 1920.

Present Condition.—March, 1928. Elastic swelling, 2½ in. by 2 in. by ¼ in., attached to skin and to inner margin of tibia, right, over the tendo Achillis. Operation scar 3 in. long over the swelling. X-ray by Dr. Tindal Atkinson showed a shadow in this region, but not obviously calcareous. Swelling said to be increasing in size and giving mechanical discomfort.

Opinions asked as to the diagnosis and operative treatment.

Discussion.—Dr. F. PARKES WEBER suggested that the swelling might be due to a juxta-articular lipomatous growth—not to be confused with a subcutaneous lipoma.

Mr. G. E. WAUGH (President) said this swelling had the characters of a cystic hygroma. It was a congenital tumour, and gave the impression of true fluctuation, but it did not really fluctuate. There was no tumour of which that was so true as cystic hygroma. The edges were slightly thickened, but that was probably from acquired pressure, or from some slight reaction after the previous operation. It should, he thought, be removed; the operation would not be very formidable, because cystic hygromata were better defined with the naked eye when one came to remove them than would be expected from the descriptions of their pathological character in the text-books.

Mr. WHITCHURCH HOWELL (in reply) said he had been unable to get a more detailed history; there had been an attempt to take away the growth. He did not think that it was a lipomatous type of tumour, or that it was in relation to any articular or synovial cavity.

Multiple Neurofibromata of the Nerves of the Upper Extremity.

By RALPH COYTE, F.R.C.S. (shown by Dr. K. M. BOWMAN-MANIFOLD).

A. B., MALE, aged 11, was brought to hospital for swelling of the left arm, noticed for the last eighteen months. There had been no pain until a few days previously, when the arm had been wrenched accidentally during play.

The left upper arm was 2 in. larger in circumference than the right. There was a firm, oval swelling, about 1½ in. long, on the inner side of the cubital fossa, and another on the outer side of the arm, which was slightly tender. The superficial veins were enlarged. A few small, hard axillary glands were palpable. There was no loss of muscular power, and sensation was normal.

At operation the triceps muscle was exposed by a longitudinal incision on the postero-lateral aspect of the arm; it was incised and a smooth fusiform tumour was exposed in its substance. This was an enlargement of the trunk of the musculo-spiral nerve, involving the whole extent of the nerve visible at operation, from its appearance between the heads of the triceps to its division, and forming a fusiform swelling 1 in. thick for 2½ to 3 in. above the division. The radial and posterior interosseous nerves and one muscular branch to the triceps were enlarged also. A wedge was removed for section. The wound healed by first intention.

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The section, stained by Weigert's method for myelin, shows a few myelin nerve fibres. The majority of the section is loose connective tissue.

There was no sign of enlargement of other nerves than those of the left arm.

Discussion.—Dr. E. A. COCKAYNE said he did not see the patient, but would like to ask two questions. (1) Were there any pigment spots on the patient? (2) Was there a family history of neurofibromatosis?

Dr. F. PARKES WEBER said he thought there were a few definite pigmented patches of skin on the back of the boy's trunk. He thought this was of some diagnostic value, though similar pigmented areas were not rare in normal persons. He divided the main signs of Recklinghausen's disease (neurofibromatosis) into three groups: (1) multiple molluscous fibromata; (2) patches of cutaneous pigmentation, chiefly of the *café au lait* tint, though darker spots might occur; (3) plexiform neuromata and swellings connected with the trunks of nerves. In the present case, although there were as yet no molluscous fibromata, there were the other two main signs of Recklinghausen's disease present. In 1905 he (Dr. Parkes Weber) had shown a girl, aged 14, with a pigmentary incomplete form of Recklinghausen's disease.[1] When in 1926 he had shown the same patient (then aged 36) the cutaneous pigmentation was about the same as in 1905, but, in addition, there were very many molluscous fibromata of various sizes, and there was also a slightly tender swelling on the right side at the lower part of the neck, which was probably connected with a nerve trunk.[2]

Mr. WHITCHURCH HOWELL said he had noticed the pigmentation of the skin of this patient, and he regarded the condition as early von Recklinghausen's disease. He asked whether the nerve was stimulated by the electric current at the time of the operation, also whether each of the nerve fibres was stimulated independently of the trunk. The child had definite atrophy of the hand, and he asked whether, as these cases usually became worse, it would be justifiable surgery to remove that 3 in. of the nerve, and form an end-to-end suture afterwards.

Dr. ELIZABETH O'FLYNN said she noticed that the child had distinct weakness of the left side of the face, which suggested that the condition was not localized to one side, and that the cranial nerves were involved. The condition was probably more generalized than was now apparent.

Mr. G. E. WAUGH (President) asked what were the earliest diagnostic evidences of von Recklinghausen's disease. He thought these were cases of fibro-neuromata. Probably the nerve when exposed at the operation looked like a piece of seaweed with nodes upon it. In these cases the nerves were very large and glistening. The branches, usually so small that they could not be seen in the dissecting room, were, in this disease, evident macroscopically. He recalled a case in which the superior gluteal nerve was attacked, and the boy came badly crippled and limping; he was also leaning towards the affected side. The diagnosis was not made beforehand. A variety of deep-seated tumours were thought of. He found, after removal of the nerve through the buttock, by palpation of the pelvis through the great sciatic notch, that several nodules had already appeared on the other branches of the lumbar and sacral plexus. For a year he had kept in touch with the boy, who still had vague pains, but no other nerves were found to have these swellings. It appeared from Alexis Thomson's monograph on the subject that these patients tended to die from involvement of the sympathetic plexus. He asked whether, in this case, there had been spread of the condition to any other nerve trunk since he was operated upon. The cases he had seen had been characterized by extreme pain, and it was difficult to know how to relieve that pain when such an important nerve as the musculo-spiral nerve was involved.

Dr. BOWMAN-MANIFOLD (in reply) said that there was seen to be a diffuse enlargement of the musculo-spiral nerve as far as it could be exposed. There was no family history of a similar affection, and she did not think there were any pigment spots. The boy had not attended the out-patient department since his discharge from the hospital. There was very little pain about the swelling, though it was somewhat tender when touched or pressed.

[1] F. Parkes Weber, "A Case of Unusual Cutaneous Pigmentation," *Brit. Journ. Dermat.*, London, 1905, xvii, p. 226.

[2] F. Parkes Weber, "Recklinghausen's Disease," *Proc. Roy. Soc. Med.*, Section of Dermatology, 1926-1927, xx, p. 22.

Section of Laryngology.

President—Mr. H. S. Barwell, F.R.C.S.

[*March* 2, 1928.]

Sinusitis in the Etiology of Mental Disorder.

By T. C. Graves, F.R.C.S., and F. A. Pickworth, M.B.

(*Introduced by Dr. P. Watson-Williams*).

I.—Mr. T. C. Graves.

(*Chief Medical Officer of the Birmingham Mental Hospitals.*)

The literature on the subject shows that sinusitis has been found in association with many varieties of mental disorder.

A large proportion of these cases are from American sources; of those described in British medical literature, only those reported by Gay French, MacGibbon and myself appear to have been certified. Under the guidance of Mr. E. Musgrave Woodman, who has been the honorary consultant officer to the Birmingham Mental Hospitals, there has, since 1924, been routine investigation for ear, nose and throat disease amongst the patients. Mr. F. D. Marsh, F.R.C.S., and later, Mr. W. Stirk Adams, F.R.C.S., have been the visiting surgeons. Dr. F. A. Pickworth has carried out the pathological and bacteriological investigations.

Sinusitis has been found on admission and at autopsy in cases associated with many types of mental disorder.

I bring forward five cases to show the association of these conditions and to demonstrate the improvement in the mental state following treatment of the sinusitis.

Description of Five Cases of Certified Mental Disorder and Nasal Sinusitis Referred to in the Paper.

Case I.—F/437. F. E. P., female, single, clerk, aged 27 on admission to mental hospital October 8, 1925. First certification.

History.—Family: healthy. Personal: at 10, severe septic scarlet fever followed by bilateral otitis media. Pneumonia at 22 and later syphilis insontium, primary sore on lip.

Present Attack.—For a year preceding admission suffered with "gastric catarrh," severe pains in stomach and "asthma." Onset of attack of mental disorder sudden, commenced with exaltation, persecutory ideation, memory poor, confusion, loss of appetite, insomnia and later violence, and noisy, uncontrollable conduct.

Certified October 8, 1925.

On Admission.—Continuation of similar conduct by day and night. Destructive to clothing and bedding. Complete confusion. Wet and dirty. Carious teeth. Vaginal and fœtid ear discharges. Spitting pus indiscriminately. Wassermann in blood, 20 units. Antiseptic treatment of cervicitis, etc. Gradual removal of carious teeth and roots.

December 16, sufficiently amenable to be seen by Mr. F. D. Marsh, F.R.C.S., Visiting Ear, Nose and Throat Surgeon, who found: Right chronic suppurative otitis media and left chronic catarrhal otitis media. Pus and polypi both middle meatus. Puncture of right antrum: pus. Bilateral Caldwell-Luc advised. Doubt felt as to

whether she would tolerate operation. Further general treatment continued. Anti-streptococcal serum given, catamenia reappeared with mental exacerbation once but ceased when serum stopped.

March 3, 1926, general condition improved so Caldwell-Luc performed by Mr. Marsh, on left antrum. Pus on culture gave St. albus and Str. subacidus. Collapse followed operation. Slow recovery. Confusion cleared. General physical and mental improvement. Menstruation returned, no associated mental exacerbation.

Able to go on parole with relatives. Left ear discharge diminished. Right ear still discharged pus. Advised to have Caldwell-Luc on right side. Accepted advice willingly, showing insight into her case. Novarsenobillon given.

May 26, 1926, right Caldwell-Luc, removal of inspissated pus, polypi and intra-nasal drainage. Recovery uneventful. Catamenia continued to appear. General physical and mental improvement. Right ear discharge diminished, became serous and fœtor disappeared.

Learnt to wash out her own antra. Discharge from cervix uteri ceased. Sent out on trial to care of relatives July 8, 1926.

Doctor's, relatives and visitor's reports satisfactory. Discharged recovered August, 1926. Attended out-patient department Ear, Nose and Throat Hospital on account of right ear. Subsequent reports as to mental stability satisfactory.

Case II.—6356. I. R., female, single, aged 19 on admission to mental hospital, July 13, 1926; first certification.

History.—Mother a physical and mental wreck, but never certified. Father, nasal catarrh. Two brothers and sister, nasal disease. At 3 patient had scarlet fever, with left otitis media. Discharge continued until 8 years of age, then lessened, but recommenced in her teens. Showed aprosexia of Guye. At age 18½ ear discharge became more profuse, and she had to leave work. Six months later, December 20, 1925, admitted to General Hospital for severe earache, otorrhœa and deafness. Simple mastoid operation. Convalescence normal. Was to have returned to hospital for treatment of recognized sinus disease, but refused to do so; became depressed; improved during May, 1926; went back to work in June, but in July became exalted; wandered from home; later auditory hallucinations and confusion, with excitement, developed. Certified and admitted to mental hospital July 13, 1926.

On Admission.— Mental state: Hallucinated, catatonic, complete confusion, noisy, mischievous, erratic, violent, destructive, wet and dirty. Auditory hallucinations appeared to be referred to *left* side.

Course.—Menstruated for five days following admission, the acute symptoms necessitating certification were therefore coincident with premenstrual phase. Dental treatment given. Wassermann negative. Conduct showed no improvement, continued destructive and violent. Course of non-specific parenteral protein therapy T.A.B. vaccine being used, an injection every fifth day during the period from July 26, 1926, to August 30, 1926. Following menstruation in September she became more composed, reverting to a phase shown after the mastoid operation. She was depressed with ideas of unworthiness, and apparently cacosmia was present as she wrote in a letter that the "stench was horrible," and that she had no self-respect left. On the basis of the cacosmia and her depressed state ideas of reference were expressed.

On October 20, 1926, examination by Mr. W. Stirk Adams, F.R.C.S., Visiting Ear, Nose and Throat Surgeon: Tonsils, pus in right; naso-pharynx, pus on posterior wall; nose, bilateral rhinitis; ear, discharge *left* ear from a posterior perforation; transillumination sinuses, dull *left* antrum and left frontal sinus.

October 27, 1926.—Sufficiently amenable to permit, under local anæsthesia, general sinus examination, using the Watson-Williams suction syringe technique, by

Mr. Adams. Pus found in left antrum and sphenoid: right antrum and sphenoid clear.

Immediately following this, an acute exacerbation of facial skin eruption occurred, and a similar eruption appeared over whole trunk and limbs, together with an acute onychia of *left* thumb (nail shed) and *left* ring finger.

November 14, 1926.—Tonsil dissection, and sinuses again washed out. Skin eruption improved considerably, and at end of December had practically disappeared from face, onychia also cleared, new nails showed no deformity but scars due to picking the irritating sores remained on limbs.

Had now gained 1 st. in weight, some mental depression still manifested but she began to show interest, especially in music, but catamenia were still and had been in abeyance since September, although aloes and iron and ultra-violet light had been given. In February, 1927, menstruation returned and continued regularly. The nose still continued to show sepsis, and on March 13, 1927, intranasal drainage of both antra was performed; following this the ear discharge diminished.

A more definite physical and mental improvement now followed. Was sent out to care of relatives in May, and after a month at home her doctor's, relatives and visitors' reports were satisfactory and she was discharged "recovered." She attended at the General Hospital for further treatment of left ear.

September, 1927, her brother reported she was back at work and doing well. February, 1928, sister reported doing well in every way.

Case III.—E. S. R., female, married, aged 37 on admission on February 7, 1927. First certification.

History: Family.—No psychotic history. *Patient.*—Childhood normal. Became a forewoman in machine shop where she did well: later married. Two children. No miscarriages. Influenzal colds not severe enough to require stay in bed. Not alcoholic.

Present Illness.—Had been working hard looking after her family. Teeth had been getting bad. During month preceding admission lost weight. Became depressed, dreamy and apathetic. Acute mental symptoms developed a week before admission and at the time when severe influenza epidemic was rampant. Became confused, visual hallucinations, terrified, talked nonsense loudly, sleep bad last four days, mistook identities, struggling and violent.

On Admission.—Complete confusion. Apathetic, depressed. Speech a rapid inconsequent muttering. Apparently the subject of auditory and visual hallucinations. Resistive to nursing attentions. Impulsively violent to those around her. Seriously ill. Phlyctenular conjunctivitis (left). Heart dilated, sounds soft and poor quality, mitral systolic murmur, regular feeble pulse 82, later became 100 to 120. Temperature normal. A few scattered rhonchi in lungs. Tongue dry. Ulcerated septic gums. Superficial ulceration soft palate, especially left side. Bilateral ptosis. Wassermann negative. Uterus partially retroverted, chronically subinvoluted, and from cervix a thick, curdy mucopurulent discharge.

Course.—Antistreptococcal serum given: Septic roots and carious teeth extracted at intervals. Gynæcologically treated with antiseptics.

At the end of February had lost half a stone, but general physical and mental state showed some improvement; later, weight was gained but she was very feeble and still confused.

Examination by Mr. W. Stirk Adams, F.R.C.S.—Tonsils: pus in both; post-nasal space: pus on left side; ears: both drums showed scar of an old healed perforation.

Transillumination of Sinuses.—Right antrum: dim; left antrum: black; frontals: clear.

Anterior Rhinoscopy.—Wet, septic nose. Still very ill, and general treatment was

carried on; bowel lavage; ultra-violet light and tonics, together with aloes and iron. Menstruation had been in abeyance since admission. Blood-pressure, 114/78, recumbent.

On July 3, 1927.—General sinus examination by Mr. Adams, using Watson-Williams suction syringe technique, gave:—Sphenoids, right, mucopus: left, clear. Ethmoids, right, clear; left, mucopus. Antra, right, clear; left, pus escaped under pressure. Left antrum drained intranasally.

Bacteriological Findings.—Right sphenoid: B. Hoffmann, St. aureus, Str. fæcalis; Str. infrequens. Left antrum: St. albus, Str. equinus. Left ethmoid: St. albus.

Following operation showed some definite physical improvement, said her head felt clearer, but she was still, though less, confused. Now able to be up and about. Catamenia returned in August. Slow physical and mental improvement; able to go out with friends. Gained weight, but sleep was variable. At times, impulsive behaviour shown.

Although tonic treatment continued, catamenia missed in September and mentally she appeared stationary; auditory hallucinations were evidently still present. Apparently did not recognize her children.

Towards end of September three injections of T.A.B. were given intravenously. A good reaction was obtained, temperatures up to 104·6° F., herpes around lips and nose, and such severe pain in back that no more injections given. From thence onward a definite mental improvement ensued.

A review by Mr. Adams on November 18, 1927 showed:—Tonsils, septic; rhinoscopy, both nasal passages clear. Left antrum (on irrigation), a few flakes of mucopus only.

Catamenia returned in October and further physical and mental improvement was noted. A gain in weight of two stones on admission. Colour good; blood-pressure, recumbent, 132/80. Eyes clear; only a few dilated vessels in left conjunctiva.

Mental State.—Conduct, manner, conversation normal. Insight gained into her case, was able to discuss and describe her auditory and visual hallucinations without slightest embarrassment. Was very grateful for the treatment she had received and able to explain the effects, e.g., that her mind became definitely clear a week after the T.A.B. treatment and that at the last menstruation she had a "moving-about sensation" in her head and after the completion of the period her head felt clearer.

She was sent home in care of relatives and after a month was discharged "recovered" as reports were satisfactory.

Case IV.—H. D. O., male, single, aged 20. Admission to mental hospital, March 8, 1927, first certification.

History.—No psychotic family history. Always a steady worker.

Present Illness.—Indefinitely ill for eight months before admission, lost weight. Influenza six weeks, mental symptoms a week before admission. Sat alone in the dark, lost interest, insomnia, heard imaginary people making noises outside. Exalted, grandiose ideas, talkative, restless, laughing and crying.

On Admission.—Confused, irresponsible, exalted, suspicious. Muttering. Vivid auditory and visual hallucinations. Flights of ideas. Grimacing and attitudinizing, rolled his head about, turned it more constantly to left. Food refusal, spoon-feeding and constant attention required. Tonsils enlarged. Carious teeth. Bilateral ptosis more on left. Wassermann negative.

Course.—Abscessed carious teeth removed. Radiogram of sinuses, clear. He remained as before but weight rose. Anti-streptococcal serum produced no improvement. Later his weight fell.

April 27, 1927.—Examined by Mr. W. Stirk Adams, F.R.C.S., showed ears,

normal. Antero-rhinoscopy, chronic atrophic rhinitis of mild degree. Trans-illumination, nasal sinuses: antra both dim, right more than left; frontals equally dim. Naso-pharynx, mucopus posterior wall, adenoids. A general nasal infection was present extending to pharynx and fauces.

On May 8, 1927.—General sinus examination by Mr. Adams, using Watson-Williams' suction syringe technique, gave evidence of infection of left antrum and right ethmoid. Right sphenoid not entered. Both ethmoids somewhat dense. Adenoids removed, tonsils dissected.

Bacteriological Reports.—Right ethmoid, B. alkaligenes, B. lactis aerogenes, and a large Gram-negative coccus. Left antrum: Strep. ignavus.

After recovery from operation began to talk sensibly, grimacing and attitudinizing ceased, became amenable to nursing. Ultra-violet light now given.

June 17, 1927.—Much improved, worked in grounds, well orientated, conduct normal. Some left ptosis still present.

July 9, 1927.—Weight 10 st. 1 lb. Feels well, looks well. No hallucinations. conduct normal, works well and shows initiative and interest. Gained insight into illness and stated when he heard the "voices" they appeared to be in the left ear.

July 14, 1927.—Discharged "recovered."

Case V.—W. H., male, single, 19 on admission to mental hospital on November 14, 1927.

History of restlessness and violence, crawling on floor, resistive to nursing, refused to answer questions. Duration of symptoms given as a week.

On Admission.—Futile and foolish, grimaced and giggled, attitudinized. Head and eye movements to right. Completely confused. No reply to questions. Apparently vivid auditory and visual hallucinations. Insomnia, noisy at night. Wet and dirty. Spoon-fed. Peripheral circulation poor. Blood-pressure 150/100 recumbent. Sphincters at times not controlled. Wassermann negative.

November 23, 1927.—Examination by Mr. W. Stirk Adams, F.R.C.S

Diagnosis.—Generalized sinus infection, with signs of a right subacute otitis media.

December 7, 1927.—General sinus examination, using Watson-Williams' suction syringe technique. Sphenoids, both dense, not entered; ethmoids, right mucopus, left too dense to enter; antra, both inspissated pus. Intranasal drainage of right antrum; left antrum too dense to drain—washed out completely.

Bacteriology of Sinus Wash-outs.

R. Ethmoid.—(1) Film of mucopus—Contained vast numbers of pus-cells, most undergoing degeneration; large numbers of diplococci and a few bacilli showing polar bodies. (2) Culture—Staphylococcus albus, Streptococcus mitis.

R. Antrum.—(1) Film of pus—numerous pus cells, many of which were undergoing degeneration, also large numbers of organisms, including bacilli, showing polar bodies, small bacilli and diplococci. (2) Culture—Staphylococcus aureus, B. Hoffmann, Streptococcus mitis, Streptococcus salivarius.

L. Antrum.—Culture—Staphylococcus aureus, B. Hoffmann, Streptococcus mitis.

Saline fluid controls.—Culture sterile.

January 25, 1928.—Both antra were found still discharging pus, and on February 8, 1928, a left Caldwell-Luc operation was done.

In this case sinusitis had been in existence for some time prior to onset of mental disorder. The acute mental symptoms represent an acute exacerbation of the pathological process. Although a hopeless case of dementia præcox, with cataleptic manifestations, he shows some amelioration of symptoms. He now feeds himself, is

more easy to keep clean, is more amenable, is quieter and sleeps better at night. His general physical condition is improving. Blood-pressure now 140/86 recumbent.

Conclusions based on our own Observations of Cases of Mental Disorder.

Incidence of the Sinusitis.

Of the cases admitted during 1927, ear, nose and throat disease was found to be present in 50 per cent.

In half this number circumstances permitted a general examination of the sphenoid, ethmoid and antral sinuses by the Watson-Williams suction syringe method. All except one gave clinical evidence of sinus disease.

Causal Factors Common to Nasal Disease and Mental Disorder.

(*A*) *Heredity.*

(*a*) *Nasal Disease.*—We have found heredity of nasal disease in cases of mental disorder, in which heredity of insanity is not traceable.

(*b*) *Mental Disorder.*—Psychotic heredity has been found associated with heredity of nasal disease: a bad prognostic combination.

(*B*) *Acquired Disease.*

(*a*) *Influenza.*—Is a recognized cause of mental disorder. The onset of the mental disorder following influenza may be (1) immediate or (2) delayed.

(1) The "immediate" group generally shows profound toxæmia, and in some the symptoms of influenza may be overlooked as the clinical picture changes so rapidly.

(2) In the "delayed" group several months may elapse before mental symptoms are pronounced.

(*b*) *Oral Sepsis.*—Seventy-seven per cent. of our cases in 1927 showed oral sepsis.

(*c*) *Ear, Nose and Throat Disease.*—In many cases a history may be obtained of an acute infection before puberty. Others have had symptoms of long-standing chronic infections.

Some Signs and Symptoms of Sinus Disease met with in Cases of Mental Disorder.

(*A*) *General Toxæmia.*

Circulatory disturbances, with high or low blood-pressure, pallor or cyanosis, intermittent slight pyrexia, general malnutrition.

(*B*) *Local Effects.*

Conjunctivitis. Herpes. Cutaneous sepsis of face, and conditions resembling erysipelas.

(*C*) *Delusions founded on a basis of Local Nerve Irritation.*

(1) *Common Sensation*—(*a*) Direct.—"A maggot at the back of the eyes." (*b*) Referred.—Pressure, heat, burning, numbing, movements in the head: e.g., "a spell produced by someone living opposite playing on the top of the head making it hot." "A ton weight on the top of the head." "Shot running about in the head." "Electric battery playing on the head." "A machine in the head."

(2) *Special Sense*—(*a*) Smell: Burnt flesh, gassing, "they always keep emptying the privies near me." (*b*) Sight: Knives flashing in front of the eyes; faces in pictures were seen by the patient in Case III. (*c*) Taste: Unpleasant tastes, ideas of food poisoning. (*d*) Hearing: Noises in the head, ticking, aeroplanes, noises of a crowd, imaginary "voices."

(3) *Local Motor Effects.*—Amongst others may be noted during the acute phase rolling the head from side to side, or sometimes more to one side. Ptosis. Grimacing. Spasmodic facial movements. Fibrillary twitchings. Pupil disturbances—inequality, which may disappear on treatment of the sinus.

(*D*) *Remote Effects.*

Gastro-intestinal disorders ("asylum colitis," a sequel). Sensations of fullness at the epigastrium, sinking, feeling pain, resulting in delusions of a "hypochondriacal" character.

General cutaneous sepsis (as shown in Case II).

FACTORS INFLUENCING PERIODICITY AND RECURRENCE.

Include reproductive demands such as menstruation.

Alcohol. Climatic changes. Re-infection.

Note concerning Menstruation.

Mental disorder is often more pronounced in the premenstrual and menstrual phases. As the mental disorder becomes more profound, menstruation ceases although an exacerbation of mental symptoms may occur at the time menstruation would be due. Following treatment of a sinusitis many cases have shown, first, the return of menstruation accompanied by exacerbation of mental symptoms, later by menstruation without such exacerbation, indicating a return to normality.

EFFECTS OF TREATMENT.

(*a*) Immediate improvement: In some cases within a month substantial physical and mental improvement is noted (e.g., Cases I and IV).

(*b*) Retarded improvement: The larger number improve slowly over a period of six months (e.g., Cases II and III). Dr. Patrick Watson-Williams' case, "W. M.," is a good example of this group.

(*c*) Delayed improvement: A few cases have shown definite improvement after six months.

(*d*) Prolonged or no change: Only slight improvement after a long period may indicate that other septic foci or disease processes are present.

We have under care at present a patient whose mental symptoms improved definitely, but not wholly, after sinus disease had been dealt with, but she has remained stationary for a year. Subsequent operative measures elsewhere appear to have completed a cure. Such treatment would, however, have been ineffectual without that of the sinusitis.

On the other hand there are cases in which permanent damage has been caused by the disease process, so that recovery cannot be expected.

DURATION OF MENTAL DISORDER PREVIOUS TO TREATMENT.

In a few cases of mental disorder in which sinusitis was suspected from clinical examination the treatment was unavoidably delayed; during this period no improvement in the mental condition resulted, but it did follow later operative measures. Similarly several cases of long-standing mental disorder have shown considerable improvement after treatment of sinusitis.

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Results after Discharge.

Reports are now being collected concerning the progress of fifty cases of mental disorder treated for sinusitis and discharged.

Included in this number are patients who had improved without nasal treatment and had been sent out on trial or discharged but subsequently had to be readmitted. Treatment for sinusitis was followed by improvement and discharge with still continuing good mental health after a longer time than elapsed following the first discharge.

The reports are encouraging, for it must be remembered that we are not dealing with early cases of patients who voluntarily seek treatment but with cases of certified mental disorder and generally long-standing disease.

II.—Dr. F. A. Pickworth

(*Director of the Joint Board of Research, City and University of Birmingham*).

Abstract.—Evidence is brought forward to show the relation of acute infection of the upper respiratory tract to nasal sinusitis and subsequent local invasion of bone, pituitary capsule, hypophysis and neighbouring brain tissue (hypothalamus). This region of the brain contains practically all the nervous centres connected with metabolism. Mental activity is also a metabolic function of a higher order, and there is reason to suppose that local damage of brain tissue near the vegetative centres may also cause symptoms of disorder of the highest metabolic function, namely mental activity.

The ætiology of mental disorder has hitherto been very little understood, due to the difficulty of demonstrating definite and consistent pathological changes in the brain, the dissociation of the study of the brain from the study of pathological processes in the rest of the body, and the fact that brain anatomy and physiology and the ætiology of general diseases have only recently developed to such an extent as to render correlation possible.

Mental processes are essentially a form of higher metabolic activity, and, considered in a limited sense which does not include the memory and intellectual processes of the brain cortex, there is thus reason for assuming that the nervous structures concerned with this function are anatomically proximate to the centres concerned with general body metabolism. It is just this part of mental activity which is disordered in the psychoses.

Nearly every case of active mental disease also shows definite symptoms of metabolic disorder, as the following table shows:—

Table I.—Association of Mental Disease and Metabolic Disorder.

Maniac depressive	Dementia præcox	Epilepsy	General paralysis
Insomnia	Sudden variations of weight	Drowsiness	Obesity
Constipation	Obesity	Apathy	Marasmus
Glycosuria	Catalepsy	Pyrexia	Sudden variations of weight
Amenorrhœa	Salivation	Subnormal temperature	Trophic ulcers
Sudden variations of weight	Seborrhœa	Abnormal hunger	Pyrexia
Polydipsia	Cyanosis	Glycosuria	Subnormal temperature
Polyuria	Amenorrhœa	Water and salt metabolism (1)	Abnormal hunger
Blood-pressure variation	Retention of urine	Acid base equilibrium (1)	Polyuria

Recent work has shown that all the above metabolic disorders are due to disturbances of nervous centres located in the hypothalamus. Table II shows the nerve centres of this region.

TABLE II.—NERVE CENTRES OF THE HYPOTHALAMUS.

Control the smooth muscles of	Control the secretion of	Control the metabolism of	Symptoms of Dysfunction	Pathological changes occur in
Eye	Sweat	Sugar	Glycosuria	Diabetes insipidus
Bladder	Saliva	Protein	Albuminuria	Dystrophia-adiposo-genitalis
Uterus	Tears	Water	Polydipsia	Narcolepsy (5)
Blood-vessels	Urine	Temperature	Pyrexia	Graves' disease (5)
	Pancreatic secretion	Fat	Subnormal temperature	Encephalitis lethargica
? Stomach	? Gastric secretion	Trophic skin	Obesity	Parkinsonism
? Small intestine	? Cerebro-spinal fluid	Sex-gland activity	Sudden loss or gain of weight	? Some forms of epilepsy
Also:—		Sleep	Amenorrhœa	? Some forms of mental disease
Striped skeletal muscle (involuntary)		Mineral salts of the body fluids	Impotence	
Plastic muscle tonus (4)		? Acid base equilibrium	Insomnia	
		? Digestion	Drowsiness	
			Apathy	
			Trophic ulcers	
			Abnormal hunger	
			Catalepsy	
			Marasmus	
			Hyperhidrosis	
			Salivation	
			Seborrhœa	
			Lacrimation	
			Polyuria	
			Constipation	
			Polypnœa	
			Retention of urine	
			? Achlorhydria	
			? Cyanosis	
			? Dilatation of stomach	
			? Dilatation of small intestine	
			? Sudden lassitude	
			? Hypochondria	

It is important to emphasize here that the above functions are located in a very small circumscribed area, which is adjacent to the pituitary stalk.

The following case demonstrates that sphenoidal sinusitis may involve the pituitary capsule and stalk in the septic process, and therefore may cause pathological change in the hypothalamus by (i) septic extension, (ii) toxic products, (iii) disturbed pituitary secretion. M. L., a woman, aged 58, who developed epilepsy and mental symptoms, dying four years later. Post-mortem examination showed a perforation of the pituitary fossa from the right sphenoid sinus and chronic bone changes consistent with the period of symptoms (Specimen No. 1, Fig. 1). Microscopical investigations showed organisms in the pituitary capsule and in a section of the pituitary stalk.

This demonstrates a previously unrecognized method of invasion of the central nervous system and deserves consideration, especially as the flow of secretion is towards the brain in the pituitary stalk, whereas, apart from vascular infection the only other means of access to the brain are: extension through the dura, which is extremely difficult on account of the ingenious repair processes connected with

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this membrane; and the passage along nerve sheaths, which has been shown to occur, but, nevertheless, is rendered difficult by the centrifugal flow of cerebro-spinal fluid.

The above case showed ravenous appetite before the onset of epileptic fits, and this, with the fits and psychical disorder, shows disturbance of the hypothalamus and pituitary; it must be remembered that these have a very intimate inter-relation.

The bone changes in the above specimen have a very important significance in that they demonstrate microscopically the chronicity of an infection which may occur in this locality. Many of our post-mortem specimens show abnormalities of the pituitary (discoloration, presence of large colloid masses, enlargement of the cleft in the pars intermedia and thickening of the capsule), which makes it seem

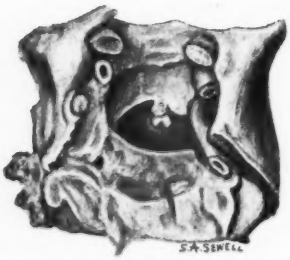

FIG. 1.

FIG. 2.

FIG. 1.—Pituitary fossa showing perforation and thick bony ridge.
FIG. 2.—Pituitary stalk, Case M.L., showing diplococci.

probable that during life microscopic but definite extensions of septic processes into the pituitary or cranial cavity occur which do not progress to abscess formation.

We must, however, not expect to be able to demonstrate histological changes in every case in which the above processes occur; firstly, because they are not immediately fatal, secondly, because the probable low virulence of invading organism and high resistance of the patient do not admit of pus or demonstrable tissue changes, such as occur in acute pyogenic infections; and thirdly, because the original infection may have completely resolved, although mental symptoms, e.g., dementia, persist on account of permanent damage of nerve tissue.

The pituitary fossa usually projects well into the sphenoid sinus (specimens 2 and 3). The glairy secretion and polypi in specimen No. 3 are indicative of chronic infection. The deleterious effect of a condition of this nature upon the pituitary

and its secretion cannot be doubted, especially if it is borne in mind that the bony walls of the sinus, once invaded, offer very little resistance to the spread of organisms.

It is only the dense fascial planes which offer great resistance to the penetrations of organisms, as the fourth specimen shown illustrates very well. The sphenoid is entirely carious, but the dura mater very much thickened and evidently for many months during life successfully inhibited the spread of infection to the brain.

Fig. 4 (Specimen No. 5) is from a case of suicide, in which the only macroscopic pathological change found post mortem was a curious polypoid condition of the

FIG. 3.—Frontal sinus illustrating small size of bacteria in comparison with a Haversian canal.

FIG. 4.—Sphenoidal sinus. Specimen No. 5.

posterior part of a large sphenoid cavity; the bone of the pituitary fossa is discoloured and there is obvious abnormality of the pituitary.

There has been one case of pituitary abscess in the Birmingham Mental Hospitals, and three similar cases have been published in the literature. In addition, Winternitz described septic involvement of the pituitary fossa from a sphenoid sinusitis, which, however, soon proved fatal. Ortmann described a case of septic involvement of the sella and demonstrated diplococci in the bone and meninges.

The conception of the nature of bacteriological spread in chronic conditions is not easy. Acute septic extension of organisms into the cranial cavity results in rapid

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fatal spread with much accompanying tissue reaction; more chronic infections, as in general paralysis and lethargica encephalitis, result in lymphocytic infiltration; but in the still more chronic extension from a long-standing sinusitis, we have what we believe to be an infection of a different nature. The specific organisms are very sparsely distributed and often partly digested, and surrounded by a glairy mucilaginous secretion such as was present in specimens 9 and 11. They probably are in a state of inhibition such as occurs when grown in immune sera, and it is surmised that a succession of abortive attempts at tissue invasion occurs with periods of lessened resistance and ill-health of the patient. Sinusitis must be regarded as a condition of unstable equilibrium between the patient's resistive capacity and the virulence of the invading organism.

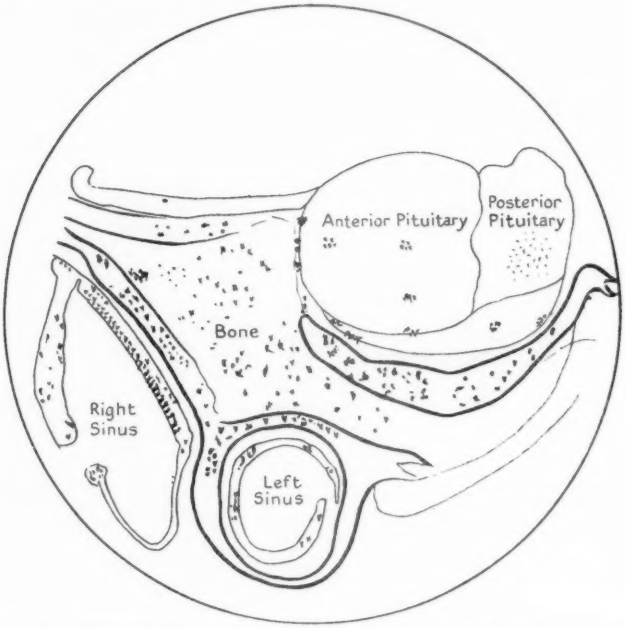

FIG. 5.—Sphenoidal sinuses of G.S. Spots indicate situation of diplococci.

With regard to the type of invading organism; we have examined 400 specimens from 103 cases but are not yet in a position to give reliable figures because control tests for sterility have been unsatisfactory with regard to air-borne organisms. The work is being continued. It may, however, be mentioned that acutely virulent organisms have rarely been found; possibly such organisms are unable to grow sufficiently in a patient whose tissues have become immune, and that less virulent varieties are able to maintain the chronic infection. We have found a large number of streptococci, diphtheroids, Staphylococcus aureus, and the type of intestinal organisms other than coli often found in the fæces of mental hospital patients.

Specimen No. 6 shows polypi surrounding the interior of the sphenoid ostium. Such a condition might occlude the ostium during life, and this brings us to the

interesting consideration of a positive gas pressure in the sinus due to bacteria; many kinds of which produce gas from sugar such is as normally present in blood (e.g., B. Friedländer). Such pressure would obstruct the blood-supply of the epithelium and such organisms as were already within the lymphatics would be distributed centrifugally: a simple explanation of the histological evidence in the case of G. S. (Specimen No. 2) in which organisms from the sphenoid sinus were seen distributed throughout the bone, in the pituitary gland and capsule (fig. 5).

Further indirect evidence is obtained from the association of influenza and mental disorder, which probably occurs through the medium of an infected nasal sinus. The following Table shows the association of infection, sinusitis and mental disorder.

TABLE III.—ASSOCIATION OF INFECTION, SINUSITIS AND MENTAL DISORDER.

Infection	Nasal Sinusitis	Mental Disorder	Observer
—	2 per cent. total population	—	Chairi
—	Many cases	Early symptoms ...	Watson-Williams
—	Many cases	Certified psychoses ...	Graves
—	50 per cent. clinical ear, throat and nose infections	Various psychoses, one year's admissions	Graves
—	65/66 of above macroscopic infections of sinus mucosa	Various psychoses, one year's admissions	Graves
—	18/28 sphenoid sinus infections P.M.	Various psychoses ...	Pickworth
Influenza	—	192 psychoses	Kleist
Influenza	—	4/2,500 soldiers ...	Fell
Influenza	—	62 cases	Gordon
Influenza	—	18 cases	Harris
Influenza	70/1534	—	Bryan Howard
Fatal influenza	25/39	—	Pearce
Fatal influenza	75 per cent.	—	Franckel
Fatal influenza	21/42 (sphenoid)	—	Prymm
Fatal influenza	64 per cent. (sphenoid) ...	—	Bruch
Typhoid fever	—	1·5 to 2·5 per cent. ...	Friedländer
Infectious diseases ...	35/360 (sphenoid)	—	Wertheim
Syphilis	15 per cent. (specific) ...	—	Gerber
Encephalitis lethargica ...	Many cases	? usual proportion psychotic sequelæ	Yates and others
Encephalitis lethargica ...	Four cases	Early psychotic symptoms	Kasanin and Petersen

The association of sinusitis and pituitary hypothalamic defect is of interest not only theoretically but practically, as it has an important bearing on prevention and treatment; a case showing acromegalic symptoms, apathy, headache and somnolence which improved considerably on opening the sphenoid, has been published.

The mental improvement which often follows treatment of the infected sinuses in Mr. Graves' series of cases is further evidence of the inter-relation of these conditions.

APPENDIX.

Description of Specimens shown, illustrating the paper.

Specimen No. 1. M. L.—Pituitary fossa seen from above, showing perforation from right sphenoidal sinus and chronic bone changes. Note especially the ridge to which the dural capsule of the pituitary was attached. The bone posterior to this was bare and the space between it and the dura full of thick, glairy, mucilaginous secretion. From a female patient, aged 62. Epilepsy with insanity, four years' duration.

Specimen No. 2.—Left: O. B.—Large sphenoid sinus to contrast with:—*Right:* G. S.—A sagittal section showing left sphenoidal sinus and pituitary fossa. Stilette is passed into the natural ostium of the sphenoidal sinus. The mucosa of the sinus is hypertrophied and the bony walls are thick, suggesting chronic infection of the sinus of long duration. Microscopically, bacterial invasion of sinus membrane, bone and pituitary (slides demonstrated). From a male patient, aged 62. Demented, epileptic, myocardial degeneration.

Specimen No. 2a.—The mucosa of the stomach of the case of sphenoidal sinus (Specimen No. 2) infection showing hæmorrhagic gastritis. The hæmorrhagic areas are evenly spaced, superficial, and resemble a leopard's skin. A cleared specimen (No. 2b) shows hæmorrhages are near the surface, and that vessels underneath are swollen and thrombosed. Histological examination shows organisms sparsely in the thrombi.

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Specimen No. 3. J. C.—Sagittal section of sphenoid sinus showing thick, glairy, secretion (much shrunken by fixative); ? polypus underneath it; injection of sinus membrane; pituitary fossa projecting well into sinus. From a male patient, aged 31. Insanity with epilepsy of seven years' duration; wild confusional excitement, hallucinated, homicidal and suicidal. 1,214 fits in hospital.

Specimen No. 4. J. L.—Sagittal section showing sphenoid sinus. Pituitary fossa, carious bone and enormously thickened dura mater. From a male patient, aged 63. Chronic melancholia of thirty years' duration. Hypochrondriacal. Severe headaches; later apathetic and demented. Wassermann negative. *Specimen 4b.*—A cleared specimen to show the caries.

Specimen No. 5. J. J.—Sagittal section of sphenoid and pituitary. The posterior part of the sphenoid sinus shows an enormous thickening of the sinus membrane. The bone is discoloured, especially between the sinus and pituitary fossa. Anterior part of sinus injected. Pituitary shows abnormal wide cleft in pars intermedia with discolouring and thickening of capsule. From a male patient, aged 38. Recurrent melancholia, headaches. After partial improvement relapsed and committed suicide. The sphenoid condition was the only macroscopic lesion found post mortem.

Specimen No. 6. A. N.—Horizontal section showing sphenoid sinuses. Right is larger than left and has a stilette passed through the ostium. Within the sinus are inflamed polypi surrounding the ostium. From a female patient, aged 27. Post-encephalitic insanity, restless, exalted, suspicious. Patient had headache of a "sharp, stinging" character, located at the root of the nose.

Specimen No. 7. A. H.—Sagittal section showing right sphenoidal sinus as a double cavity which communicated nearer the mid-line. Optic canal is seen to be projecting well into the sinus. There is inflammation, and a small polypus on the bone near the pituitary. From a female patient, aged 65, single. Recurrent mania nineteen years.

Specimen No. 8. A. M.—Sagittal section showing left sphenoidal sinus. Some evidence of inflammation. Note the large mass of transparent colloid in the posterior part of the pituitary gland. From a male patient, aged 42. Epileptic insanity of at least seventeen years' duration. No fits for a year before death; but was demented, querulous and apathetic.

Specimen No. 9. C. M.—Sagittal section showing left sphenoid sinus and pituitary. Posterior part of sinus contains polypi (considerably shrunken by fixative), some thickening of capsule of the pituitary and discoloration in the posterior part of the gland. From a male patient, aged 29. Epilepsy with insanity of eight years' duration. Dangerous, raving, screaming, or sullen and morose.

Specimen No. 10. B. S.—Sagittal section showing left sphenoid sinus. Shows a moderate sized polypus on the anterior inferior wall. From a male patient, aged 38. Insanity of sixteen years' duration. Quarrelsome, threatened to kill wife and child. Muttering, foolish laughter, hallucinated, demented. Wassermann positive.

Specimen No. 11. L. T.—Sagittal section of sphenoidal sinus and pituitary. A chronically inflamed sinus with hypertrophied sinus membrane, local injection and containing a considerable quantity of thick, glairy mucus. From a female patient aged 32. Catatonic. Dementia præcox. Noisy, excited and extremely restless. Recently much loss of weight.

Microscopical Demonstrations.

(1) Slide of G. S., showing diplococci, mostly undergoing digestion, but a few morphologically perfect, invading the mucosa of sphenoidal sinus, bone and pituitary.

(2) Slide of G. S., showing similar diplococci within thrombi of small vessels of stomach above which the characteristic leopard's skin hæmorrhages are present.

(3) Slide of A. T., showing bacteria invading bone of frontal sinus.

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Discussion.—Mr. W. STIRK ADAMS said he had been associated with the authors in this work for the past eighteen months, and had examined 480 patients selected by Mr. Graves and his staff. In 329 he had found evidence of sepsis in the nose. He included in this group all patients who had pus, crusts, excessive mucus, or mucous polypi in their nasal passages, or a granular pharyngitis, and also those who, on transillumination, showed dimming of the antra.

In 177 patients the Watson-Williams method of examination of sphenoid, posterior ethmoid, and antra was carried out, and in 161 of these there was evidence of infection. The sphenoid was affected either alone, or with other sinuses, in 78 patients, the posterior ethmoid in 106, the antra in 120. These figures were based on clinical findings alone—pus in the syringe, a thickened velvety mucosa, or, in a few cases, polypi withdrawn on the antral cannula. A considerable number of the washings had been examined by culture, but the results of these had not been included.

In relation to the chronicity of the infection in these patients, the presence of changes in the bony structure of the nasal walls of the sphenoid and ethmoid had attracted his attention. Osteosclerosis was noted in fifty-one of these patients, and in forty-two he had failed to enter one or more posterior sinuses, the number of individual sinus failures being sixty-three. He considered that osteosclerosis was more frequently met with than osteoporosis.

Mr. T. B. LAYTON said that there was no branch of surgery in which a more sceptical approach was needed than that of sinusitis. He would not have the reader of the paper and those others who had brought their data before them, think he intended any disparagement in what he had to say, and he hoped they would continue in their work; but an agnostic standpoint was justified on the subject. He would have it understood that he criticized his own technique just as severely as he did that of others.

The first point to which he would draw attention was that in none of these cases had the diagnosis been made by the fundamental routine way of finding pus within the nose, that is by thoroughly cleansing the nose until all had been removed, and of then waiting to see if it returned. In each of these cases it was made by less sure methods, by the use of the cannula, by the cultivation of washings, and by transillumination.

Secondly, he wished to point out the remarkable bacteriological results which had been obtained. No single organism had been found, but a multiplicity, and of a type not usually associated with suppurative pathological conditions: particularly was it noticeable that hæmolytic streptococci were absent from all these cases. He felt they were justified in saying that these were not typical cases of suppurative sinusitis. Some Members would remember that last summer (1927) he had read a paper[1] in support of the view that there was a distinct type of sinusitis entirely separate from the suppurative, and that further investigation was necessary to discover whether this was the case. In the meantime he wondered whether the cases described to-day might come under this category.

The great difficulty, however, was to distinguish between a local inflammation inside the cavity of the sinuses and a general inflammation of the whole mucosa of the nose, of which the lining of the cavities was a part. Under these circumstances the condition should be classed as a rhinitis rather than a sinusitis. External dirt would often cause such a state; one saw it frequently in children when working in a poor and overcrowded district, and he wondered whether the condition found in the nose of these patients was the result of external dirt. It might be remarked that every case was in a dirty condition when admitted, apparently with the exception of Case 4, in which there was a potent source of superficial infection in the form of carious teeth. If such was the explanation of these cases, then the improvement within the nose might have resulted from the general improvement of the patient.

He wished particularly to speak of the "Watson-Williams technique" for investigating sinuses. Nine years ago Dr. Watson-Williams had brought his method before the Section,[2] and he (the speaker) made certain criticisms of it. Since that time he had watched Dr. Watson-Williams use it at Bristol and at Manchester, and Mr. Eric Watson-Williams had demonstrated it in his (Mr. Layton's) theatre. Nine years' work had not given him any reason to reverse his criticisms. He had then said that "he did not doubt Dr. Watson-Williams' rhinological technique, but he did doubt his bacteriological technique." On this subject of bacteriological technique he would point out that the general surgeon with his so-called aseptic technique, was far behind the bacteriologist in his work. To realize this one had but to consider the number of times material sent to the laboratory for investigation turned out to be useless, because it had become contaminated with some adventitious organism. Inside the nose one was in a greater difficulty than on the outside of the body. In exploring the sphenoidal sinus by the Watson-Williams technique, a blunt trocar within a cannula was passed into the nose, touching the septum or middle turbinal, and was then driven through the anterior wall of the cavity; the trocar was then withdrawn through the cannula, saline was injected and sucked back again. It was impossible to say that the organisms so grown came from the anterior of the sphenoid sinus, and not from the mucous membrane covering its anterior surface or from some other part of the nose. So far as the posterior ethmoidal cells were concerned, he now suspected that even the rhinological technique

1 *Proceedings*, 1927, xx (Sect. Laryn.), 81.
2 *Proceedings*, 1919, xii (Sect. Laryn.), 26, 27.

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of this method was at fault. In many cases when the cannula was put from underneath through the posterior end of the middle turbinal, its point lay not in an ethmoidal cell, but in the superior meatus of the nose. If in such a case mucopus were sucked up, the diagnosis of an inflammation of the cells was made whereas it was really a localized cellulitis.

With regard to the specimens shown, he was not prepared to give an opinion on the condition of a mucous membrane when looking through a piece of glass, but it was often difficult in the post-mortem room to decide whether the lining of the sphenoidal sinus was normal or inflamed, and he was not prepared to accept all the descriptions given. He could be more dogmatic with regard to one specimen, in which it was suggested that the osseous tissue round the small sphenoidal showed evidence of osteitis. Considering the variations in the size of the sphenoidal sinus and in the density of the osseous tissue of the body of the sphenoid, he did not think there was anything there that might be found in a normal sphenoid in which there was no suspicion of inflammation.

Dr. PATRICK WATSON-WILLIAMS said that at the meeting of this Section in May, 1916, he had introduced the subject of " The Influence of Nasal Sinus Infection upon the Moral and Legal Responsibility of the Patient ; " and he and other Members had then described a number of cases. Impressed with the importance of this subject he had again dealt with it nine years later in his Semon Lecture. Hence he had been gratified to find that, in the meanwhile, similar work had been carried on at Birmingham Mental Hospital by Mr. Graves and his staff and by Dr. Pickworth ; also at Trenton, New Jersey, by Dr. Henry Cotton and by others. Mr. Stirk Adams had undertaken the investigation of cases for Mr. Graves from the laryngologist's point of view. One of the diagnostic methods used by Mr. Adams was the suction process associated with the speaker's name.

He hoped Mr. Kisch would elaborate the mental aspects of his case recently shown at a meeting of the Section, in which the sphenoidal sinus infection was proved by the method which Mr. Layton deprecated. That patient, as was so often the case, recovered perfectly, almost immediately after the sphenoidal sinus had been drained.

The profession was learning much from what was being done in mental hospitals in the advanced study of mental disorder ; might one not hope for similarly beneficial therapy in relation to some forms of crime ? This might at times take the place of purely punitive measures, which, indeed, our grandfathers thought right even in cases of lunacy.

Specimens in which the stomach was also involved had been shown by Dr. Pickworth, and one showed that apparently infection was present in the blood-stream, while involvement of the hypothalamus probably had much to do with some of the symptoms.

He regretted that it was impossible to occupy time in replying to Mr. Layton as to the way of using the cannula and syringe in diagnosis ; he could only ask Mr. Layton to read more carefully the directions for the technique which the speaker had described and published on previous occasions (e.g., *Journ. of Laryng., Otol. and Rhin.*, Mar., 1912 ; *ibid.*, Oct. 1921 ; *ibid.*, Dec. 1925).

Dr. J. R. LORD said he had tried, without much success, to get ear and throat specialists to take an interest in psychiatry, and he hoped the outcome of this meeting would be a move in that direction. As had been said, the Birmingham Mental Hospitals had provided a fine example of the value in mental cases of a thorough examination for the presence of focal sepsis and its prompt removal. For long psychiatrists had been trying to separate out from the great mass of undefined mental disturbances, types or syndromes approaching disease entities having a definite causation, pathology and prognosis, symptomatology and lines of treatment. There had been separated out certain prominent types to be met with every day—manic depressive psychosis, dementia præcox, general paralysis, arterio-sclerotic psychosis, involution melancholia, senile decay, etc. There still remained, however a large number of undefined cases whose symptoms revolved round these defined types. They presented an unexplored territory, but he was pleased to say that, with the co-operation of other branches of medicine, there was a prospect of being able in the near future to separate out another definite psychosis. He did not know what it would be called, but he would strongly object to it being termed " septic psychosis."

On visiting a mental hospital one could not fail to be impressed, even on only casual examination, by the fact that the physical condition of chronic cases suggested involvement of the basal ganglia. In the early cases, on the other hand, abnormality in feeling tone was

usually the primary condition. It was an old idea that all psychoses had a prodromal or early affective disturbance, and modern biological conceptions in psychiatry supported that view. It was now generally recognized that the infra-granular layers of the cortex, the thalamo-striate level, and the machinery of the vegetative nervous system, were associated with the dynamic of mind, i.e., instincts and feeling tone. Thus there were these two parallel facts: that the chronic case showed physically definite implication or degeneration of basal structures, while in the recent and acute cases these structures were apparently also involved in that affective onset he had just described. The clinico-pathological aspects of many cases of both defined and undefined mental disorder suggested a septic or toxic causation. There could be no doubt from this point of view, that there were two groups of cases: the acute infection case, in which there was a mass invasion of anatomical areas of the brain through arterial or lymphatic channels, and another type in which a virus or toxin probably elaborated in the intestines, invaded functional levels of the nervous system by a selective action. His experience gave strong support to the hypothesis which had been advanced at this meeting of the importance of sepsis in the ætiology of mental disorders. Medical officers had brought before him mental patients for discharge, but with whom the speaker was not satisfied. Their complete recovery seemed to be delayed for some reason or other, and it had been found that a septic focus was standing in the way. He had not yet met with such a case in which effective eradication of such foci had not resulted in complete recovery of the patient. That was a strong clinical support of Mr. Graves' contentions. One had to be alive to this causation in the so-called recurrent mania, commonly a recurrent hallucinatory excitement. The history was often first a throat or stomach condition, possibly ulcerative. Then in two or three years' time an attack of wild excitement with hallucinations and considerable confusion. This cleared up, but two or three years later the same mental symptoms recurred. One patient had had six such attacks. One such attack he observed was associated with congestion and malar twitching of one side of the face, a narrowing of the orbital fissure, and excess lachrymation on that side. The twitching of the lower eyelid was from side to side—vertical and not horizontal.

It must not be supposed that treating sepsis would cure all cases of mental disease: it would not. But what was going to happen by this new line of research was to bring within the range of practical psychiatry another definite group of that still very considerable psychiatric material which was yet undefined and treated on empirical lines only. Further, a fresh light was also going to be thrown on the causation of the defined psychoses in many cases, and what was still more important, a new implement of prophylaxis forged.

Dr. WILLIAM HUNTER said that the part played by the sinuses in connexion with mental disorder was very important to the profession; actual details might well be left to laryngologists to work out in individual cases. The factor of sepsis had to be faced, just as that of syphilis and its large entry into diseased states had been faced. With regard to the name for the psychotic effects produced by sepsis, he must leave that to his psychiatrist friends, though he was personally inclined to give the term "septic" to a state in which sepsis was proved to be a causal factor. Psychiatry was being brought into closer touch with general medicine; he himself found that studies with regard to sepsis had given him a keen interest in diseases of the mind.

Mr. A. D. SHARP said he hoped that as a result of the papers just read, greater attention would be paid to the relationship of sinus suppuration to mental disorder. A case seemed to have been made out for a rhinologist to be attached officially to every mental institution. A patient ought not to be certified as insane until a thorough investigation of his sinuses had been made.

He might mention the case of a married woman, a devoted mother and wife, always healthy and cheerful. She suddenly manifested symptoms suggesting an oncoming mental disorder, i.e., morose, irritable, detached from her family and suspicious of everybody. This became so bad that it was feared she might do bodily harm to the children. As she had in the early stage complained of headache, he suggested that the nose might be examined. Pus was detected high up in the naso-pharynx. She was brought to a nursing home and he found pus in the left sphenoidal sinus, which he drained in the usual way. In a week she was greatly improved, and on the tenth day asked about her home, and desired to have

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her children brought to her. Improvement was uninterrupted, and in six weeks she was well, mentally and physically. This occurred a few years ago; there has been no relapse.

Dr. JOBSON HORNE said that he agreed generally with all that Mr. Layton had said. He considered that the case for nasal sinusitis as a cause of mental disorder had been very much overstated in the papers before them. The "bacteriological findings" mentioned by name a baker's dozen of different micro-organisms as responsible. Years ago he had investigated the bacteria in the healthy nose and sinuses; the organisms found were too numerous to be christened, and were not pathogenic. He also agreed with Mr. Layton that the technique employed was the weak point; any number of organisms could be obtained by that method, and to base nasal sepsis on such findings was not warrantable. If the Section were to accept the suggestion that nasal sepsis was an ætiological factor in seventy or eighty, or possibly ninety per cent. of cases in mental hospitals, then rhinology would be brought into ridicule.

Mr. E. D. D. DAVIS, referring to the 329 cases mentioned, asked how many patients there were in the asylum referred to.

Dr. PICKWORTH (in reply) said that a very careful and controlled technique was being employed for the investigation of the bacteriology of the sinuses, but as the controls were occasionally not sterile it was not yet advisable to publish it or to make important deductions. [A lantern slide was shown, which indicated the type of organism and the relative frequence of its occurrence in 400 specimens.] The exact type of organism, perhaps, was not so very important after all; especially if we follow Lowndes Yates' view that only a damaged mucous membrane, as in chronic sinusitis, allows the permeation of viruses, such as lethargic encephalitis. The specimen "G. S.," referred to by Mr. Layton as not showing osteitis, was certainly permeated by organisms easily seen in a microscopic section which had already been demonstrated and illustrated (fig. 5).

Mr. T. C. GRAVES (in reply) pointed out that the first case was not investigated by the Watson-Williams method; it was a clear-cut case of chronic suppurative sinusitis; pus and polypi were found in both antra.

He had been much interested in the multiplicity of the organisms found in these cases, which he believed to be the cause of the mischief; the ravages of one organism probably made way for and facilitated the destructive work of another.

The suggestion that dirt could cause sinusitis after the mental disorder had been established seemed to be negatived by the histories which, in some cases, dated back to before puberty.

The sequence of events in certain cases of sinusitis and mental disorder which had been treated was: Sore throats or infectious disease, such as scarlet fever, with subsequent chronic nasal and/or aural sepsis, later gastro-intestinal disease, then an attack of mental confusion which might temporarily clear, but which would recur and necessitate certification.

Three of the cases had not had surgical interference until nearly six months after admission, and they did not improve until surgery had been carried out. In the meantime other lines of treatment had been followed which had given good results in other cases.

He was sure these cases would not have improved as much as they did unless the sinuses had been treated; they might have stabilized at a certain level, but they would not have been able to resume family life.

With reference to the request for statistics, he preferred to give them on the basis of the annual admissions. In 1927, of 300 cases admitted to the mental hospital, 50 per cent. were considered on admission to have sufficient disease of the nose, ear or throat, to require examination by Mr. Adams, to whom they were sent, and he agreed with the diagnosis. In half these cases, i.e., 25 per cent. of the admissions, the Watson-Williams suction syringe method was carried out, and it gave evidence of sinus disease.

He wished to thank his colleagues on the visiting and resident staffs of the hospital for their observations on and treatment of the cases he had submitted.

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JOINT DISCUSSION No. 6.

Sections of Tropical Diseases, Dermatology, and Comparative Medicine.

Chairman—Dr. J. M. H. MacLeod, President of the Section of Dermatology.

[*January* 25, 1928.]

DISCUSSION ON TROPICAL MYCOSES.

Dr. J. M. H. MacLeod (Chairman).

Our knowledge of the fungi and fungus diseases, whether in tropical countries, or in temperate zones, is still far from being complete. So far as the tropical mycoses are concerned it is very imperfect. During the last twenty years, in connexion with the Hospital for Tropical Diseases, I have seen a number of cases of mycoses of tropical origin, and in one or two instances have been able to isolate causal fungi, which differed from those with which we are familiar here, but these have been exceptional.

I have been overpowered in an attempt to absorb the long and complicated classification of fungi, by Dr. Castellani, which has recently appeared in the *Archives of Dermatology and Syphilis*. It seems to me that what is needed at the moment by the physician is a simplification, rather than a multiplication of variants of different species, based on slight and uncertain differences in sugar reactions, or appearances in culture.

From the practical point of view one of the most striking facts is that similar clinical appearances can be produced by different fungi. For example, it is often impossible to prophesy the fungus which will be found to have caused some lesion of ringworm. It is sometimes difficult to tell even if a lesion is the result of such different ringworm fungi as microspora and trichophyta. Recently, considerable interest has been aroused in the dermatological world in connexion with the pathogenic, yeast-like fungi. The monilias have been found to cause not only thrush, but occasionally lesions almost indistinguishable from an epidermophyton infection. The most recent addition to our knowledge has been in connexion with the pityrosporon of Malassez, which we have now succeeded in cultivating, both in solid and fluid media, and by inoculating it, producing lesions identical with what we have long known as seborrhœic dermatitis.

[The Chairman demonstrated by means of the epidiascope a series of photographs of cutaneous mucoses, and preparations of pathogenic fungi.

(1) Specimen of *Monilia pinoyi*, from a culture grown on maltose agar.

(2) Monilia infection of the web of the fingers, in which the skin presented the white, thickened, sodden appearance, familiar in connexion with epidermophyton infection.

(3) Monilia infection of the nails, showing the pad-like puffiness and thickening of the posterior nail wall.

(4) Trichophyton infection of the nails, for comparison.

(5) Specimen of pityrosporon of Malassez, from a culture on maltose agar, showing the typical spores.

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(6) Specimen of pityrosporon of Malassez, from a culture on peptone and oleic acid showing non-septate hyphæ.
(7) Specimen of *Trichophyton rosaceum* from the beard.
(8) Patch of ringworm on the back of the hand of a boy, due to infection from a canary.
(9) Photograph of two canaries infected with ringworm].

Mr. J. Ramsbottom (British Museum, South Kensington).

So many fungi have been reported as concerned in cutaneous mycoses that a discussion of them in the time at one's disposal would amount to little more than a list of fungal names. It has seemed best therefore to consider some general biological points and to discuss recent work on fungi of the *Trichophyton* group, many of which fall within the limits of our subject.

Cultures of fungi reputed pathogenic to man are frequently sent to me, and I find it impossible to name them with the aid of the standard English work (Castellani and Chalmers' "Manual of Tropical Medicine"). Since the fungi are sent for identification the assumption is that it is impossible to recognize them from the lesions. For instance, the description of *Trichophyton* in Castellani and Chalmers' "Manual" (p. 995, ed. 3) may be taken: Trichophytoneæ with mycelial filaments and spores present in the lesions and conidial-bearing hyphæ in cultures, only attacking hairs and entirely of human origin. Almost never pyogenic.

The other generic diagnoses are similarly dependent upon some parasitic character. One must admit that there are certain groups of fungi, e.g., mildews, rusts and smuts, in which parasitism is so pronounced that up to the present it has not been found possible to grow them apart from their host plants, but cultures of these if obtainable would occasion little difficulty in recognition. On the other hand, however, it is rarely possible to recognize medical fungi from their description.

Sabouraud's classical work is the basis of our knowledge of the *Trichophyton* group, and any suggested alteration in his generic names will be strenuously opposed. Even so eminent a mycologist as Vuillemin has recently said that any interference with Sabouraud's system is, from a medical point of view, a retrograde step. The difficulty might be met in a simple way if a lesion could be recognized as due invariably to some particular fungus, even if it were necessary to confirm the identity of the fungus by growing it on some standard medium: then it would resolve itself into a "spot" diagnosis on the part of the medical investigator—the mycological systematist being left to classify the organisms in whatever way he considered advisable. Recent work has shown, however, that many cutaneous mycoses can be produced by widely differing fungi. Further, most of the fungi concerned can be grown on culture media, and they pass from laboratory to laboratory. The point arises, therefore, whether a general account of the gross appearance of a culture is a satisfactory method of description. My opinion is that it should not be impossible to draw up generic and specific diagnoses which would enable these numerous fungi to be distinguished. This is a comparatively easy matter with genera like *Debaryomyces* and the other true yeasts, and though specific distinctions are sometimes troublesome in *Aspergillus*, *Penicillium* and similar forms, the genera are well marked and easily recognizable. In the *Trichophyton* group, however, we meet with difficulty. It is common among fungi for more than one spore form to occur. The most frequent example of this is where both a conidial stage and a fruit-body are present, as, e.g., in *Aspergillus*, where the conidial form is a head bearing numerous chains of spores, and the fruit-body or perfect stage is a closed structure (*Eurotium*-stage) containing asci enclosing spores. Other fungi possess more than one conidial stage, and this is so with several of

the Dermatophytes. The matter is complicated here, however, by the phenomenon called "pleomorphism" by Sabouraud; the fungus changes in culture, loses its spore-producing properties and continues in this condition even when again inoculated into its host. Sabouraud devised two media, one for growing the fungus for examination, the other for preserving it in its original form. While agreeing that it is advisable to have a standard medium for comparing the growths of different fungi I think that Sabouraud's medium is not the best that can be found. I have had a number of these fungi in culture and am not yet convinced that, from a purely mycological standpoint, growth is satisfactory. It is annoying when a culture of a fungus is given to one to find that, as it has been grown on a certain medium, it has lost most, if not all, of its specific characters, and that these can never be regained. I would appeal to any who are interested in these fungi to obtain a little information about the possibility of growth, and, if successful, the characters of the growth on some of the usual media of mycological laboratories. It would be of great scientific interest to establish beyond doubt that pathogenic fungi can be altered gradually and permanently in culture.

During the last few years there have been three important papers on the *Trichophyton* group—Ota and Langeron (1923),[1] Grigoraki (1925)[2] and Nannizzi (1926).[3] The first two propose reclassifications of the group. To explain these classifications it is necessary first to consider some of the scientific terms used. In general mycology it has become the custom to use the term "spore" indifferently for the reproductive structures of most fungi, the "conidium" being regarded as a spore associated with the imperfect stage. For classification, however, it is important to distinguish between the different ways in which spores are formed. For the spores of perfect stages, ascospores, basidiospores, and so on, the distinctions are easy to draw. Until recent years but little detailed attention has been paid to the different methods of spore formation in the fungi with no perfect stage, i.e., the Fungi Imperfecti. One of the main groups of these, the Hyphomycetes, includes the majority of pathogenic fungi. Saccardo classified these on an artificial system which has been of the greatest value both in identification and in the coördination of results. Although it is followed by the majority of mycologists it is certain that some different system, based on natural relations and polymorphism, will have to be adopted. The problem is to gain an insight of relations where only part of the life-history is known. The veteran mycologist Vuillemin has been the main worker in this field but unfortunately has published only a series of papers on special points and not put forward a comprehensive scheme. This work of Vuillemin forms the basis of the treatment of Hyphomycetes in the works of Brumpt, and of Castellani and Chalmers, and in the two classifications about to be considered. He reserves the term "conidium" (fig. 1) for spores which at their first formation are distinct from the hyphæ and easily detached: a conidium may be borne on a conidiophore, or may be sessile on the hypha. An "aleuriospore" (fig. 2) differs from a true conidium in that it is not originally distinct from the hyphæ and is not set free until these degenerate: it may be terminal, lateral or intercalary and like the conidium may be sessile or borne on a differentiated sporophore. In contrast with these are the thallospore and the hemispore. Only the first of these requires to be considered. The "thallospore" is essentially a part of the vegetative structure (thallus) adapted in a secondary

1 Ota, M., and Langeron, M., "Nouvelle classification des Dermatophytes," *Ann. Parasitologie Hum. et Comp.*, i (1923), 305-336.

2 Grigoraki, L., "Recherches cytologiques et taxinomiques sur les Dermatophytes et quelques autres champignons parasites," *Ann. Sci. Nat. Bot.*, 10th sér., vii (1925), 165-444.

3 Nannizzi, A., "Ricerche sui rapporti morfologici e biologici tra gymnoascacee e Dermatomiciti," *Ann. Myc.*, xxiv (1926), 85-129.

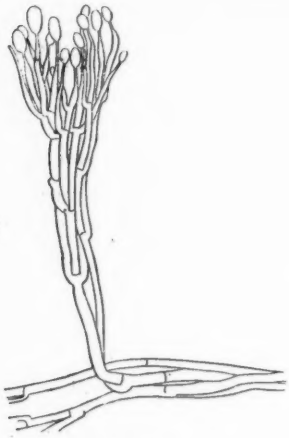


FIG. 1.

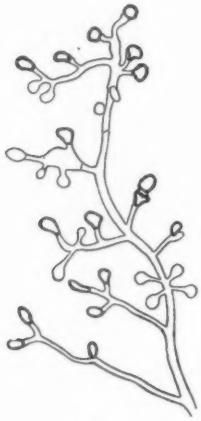


FIG. 2.

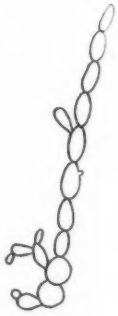


FIG. 3.

FIG. 1.—Conidia borne terminally on conidiophores (sporophores).
FIG. 2.—Aleuriospores.
FIG. 3.—Blastospores.

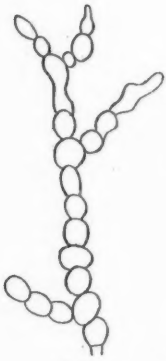


FIG. 4.

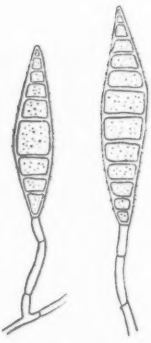
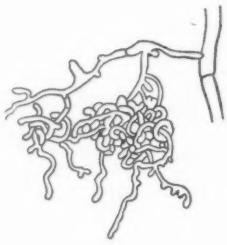


FIG. 5.

FIG. 6.



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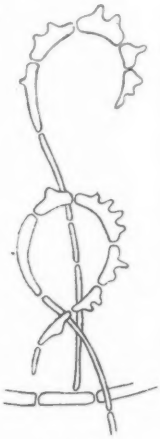

FIG. 8.

FIG. 4.—Arthrospores.
FIG. 5.—Fusiform spores (chlamydospores).
FIG. 6.—Intercalary chlamydospores.
FIG. 7.—Nodular structure.
FIG. 8.—Pectinate structures.

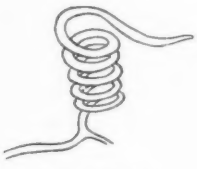

FIG. 9.—Spiral.

FIG. 10.—Clavate terminal.

manner for dissemination or preservation; thallospores include blastospores, arthrospores, and certain chlamydospores.

Blastospores (fig. 3) are spherical bodies budded off in a yeast-like manner from hyphæ or from bodies similar to themselves.

Arthrospores (fig. 4) arise from the fragmentation of vegetative hyphæ and correspond to the "oidia" of many mycologists; the hyphæ segment and form cubical or rectangular bodies which sometimes round themselves off. By encystment thick-walled chlamydospores may arise: chlamydospores may be formed also singly, either terminally (fig. 5) or in an intercalary position (fig. 6).

Ota and Langeron disregard lesions, the method of penetration, etc., in their classification which they base entirely on the characters found in a microscopical examination of cultures. The characters they rely on are indicated in the following key:—

1.	Aleuriospores numerous, well developed, isolated or in clusters	2
	Aleuriospores rare and imperfect, isolated, rarely in clusters	3
2.	No fusiform spores or accessory organs	*Trichophyton*
	Fusiform chlamydospores (fig. 5) and other accessory structures (nodular [fig. 7] and pectinate [fig. 8] spiral [fig. 9], etc.)	*Sabouraudites*
3.	Numerous and well characterized fusiform spores	*Epidermophyton*
	No fusiform spores	4
4.	Arthrospores accompanied by accessory structures (clavate terminals [fig. 10] chlamydospores, etc.)	*Grubyella*
	Arthrospores without accessory structures	5
5.	Arthrospores in chains	*Bodina*
	Arthrospores ill defined	*Endodermophyton*

The genus *Sabouraudites* is subdivided as follows:—

1.	Aleuriospores and fusiform spores	2
	Aleuriospores and nodular structures, but no fusiform spores	*Aleuramma*
2.	Nodular structures	*Closteramma*
	No nodular structures	*Aleurocloster*

The fusiform spores of Dermatophytes are of various shapes and sizes. Apparently they are to be regarded as transformed chains of chlamydospores. The nodular structures mentioned in the key are masses of interwoven hyphæ which assume various forms and sometimes simulate fruiting bodies.

Ota and Langeron retain the generic names—*Trichophyton*, *Endodermophyton*, *Epidermophyton*, the characters of the first and last being emended.

Grigoraki, on the other hand, makes a clean sweep of all the old genera, and, further, adopts none of the new ones of Ota and Langeron. This is most reprehensible in any taxonomic work and leads to tremendous confusion. It is often more simple for an author to disregard previous generic names than to emend diagnoses, but in doing so he invites a similar wholesale rejection of his own names. Because of this fault it is doubtful whether Grigoraki's work will obtain the recognition to which it is entitled. His paper of almost three hundred pages discusses the cytology of many Dermatophytes and allied fungi and details the pleomorphism of several species. As a result of his studies he bases his classification on the microscopical appearance in *initial* cultures.

His genera are:—

Closterosporia.—Numerous fusiform spores, some aleuriospores and chlamydospores.

Closteroaleurosporia.—Some fusiform spores, many aleuriospores and chlamydospores.

Chlamydoaleurosporia.—Chlamydospores and aleuriospores.

Spiralia.—Fusiform spores, aleuriospores and chlamydospores; hyphæ with characteristic spiral terminals.

Aleurosporia.—Only aleuriospores.

Arthrosporia.—Only arthrospores.

Commenting on this classification, Langeron has pointed out that some of the genera are more or less identical with those proposed by Ota and Langeron: *Closteroaleurosporia* includes only *Sabouraudites*, *Aleurosporia* includes only their *Trichophyton*, *Spiralia* is part of the subgenus *Aleurocloster*, and *Arthrosporia* is *Endodermophyton*, *Bodina* and *Grubyella*.

This multiplication of genera will no doubt serve to hamper the adoption of modifications of the former classification.

The names common to Castellani and Chalmers, Ota and Langeron and Grigoraki are listed below:—

Castellani and Chalmers.	Ota and Langeron.	Grigoraki.
Microsporon Audouini	Aleurocloster (Sabouraudites)	Closteroaleurosporia
M. umbonatum	Trichophyton	Chlamydoaleurosporia
M. lanosum	Closteramma ,,	,,
M. felinum	,, ,,	Closteroaleurosporia
M. fulvum	Aleurocloster ,,	,,
M. pubescens	,, ,,	Closterosporia
Trichophyton tonsurans	Trichophyton	Chlamydoaleurosporia
T. violaceum	Bodina	Arthrosporia
T. umbilicatum	Trichophyton	Chlamydoaleurosporia
T. effractum	,,	,,
T. Currii	Ateleothylax	Ateleothylax (Matruchotiella)
Neotrichophyton plicatile	Trichophyton	Aleurosporia
Ectotrichophyton ochraceum	,,	Arthrosporia
E. album	Grubyella	,,
E. gypseum	Aleurocloster ,,	Closterosporia
E. asteroides	,, ,,	Spiralia
E. granulosum	,, ,,	Chlamydoaleurosporia
E. lacticolor	Aleuramma ,,	,,
E. radiatum	Aleurocloster ,,	Spiralia
E. felinum	Trichophyton	Aleurosporia
E. denticulatum	,,	,,
Achorion Schoenleinii	Grubyella	Arthrosporia
A. quinckeanum	Closteramma ,,	Closteroaleurosporia
A. gypseum	Aleurocloster ,,	,,
Lophophyton gallinae	,, ,,	,,
Epidermophyton inguinalis	Epidermophyton	Closterosporia
Endodermophyton tropicale	Endodermophyton	Arthrosporia
E. indica	,,	,,

Both of these classifications propose a separate genus for *Trichophyton Currii* Chalmers and Mitchell. The describers of this species recorded the presence of a fruit-body which was assumed to contain asci. It is probable that the structure was not a true cleistocarp such as is met with in *Aspergillus* (*Eurotium*) but sterile. Ota and Langeron proposed the name *Ateleothylax* for this species and this is adopted by Grigoraki in place of the name *Matruchotiella* which he had proposed in a preliminary note.

The presence of a fruit-body of this kind makes the results obtained by Nannizzi of peculiar interest. It will be recalled that Matruchot and Dassonville in a series of papers (1895-1902) put forward the suggestion that *Trichophyton* and its allies were related to Gymnoascaceæ, a somewhat primitive group of Ascomycetes characterized by subglobose asci surrounded by a loose peridium instead of a definite wall, and recorded from soil, feathers, old dung, bird's nests, and so on. This hypothesis was based on the similarity in cultural characters particularly with *Ctenomyces serratus* (*Arthroderma Curreyi*[1]).

[1] This name is earlier than *Ctenomyces*, but owing to the small asci having been overlooked, it has usually been regarded as a Hyphomycete.

Nannizzi has made a detailed study of many of the Gymnoascaceæ and compared them with *Trichophyton radiolatum*, *T. asteroides*, *T. denticulatum*, *T. felinum*, *T. equinum*, *T. granulosum* and *Microsporon lanosum*. The Dermatophytes were grown in artificial media and also on leather, bird's wings and human hair. The first five in the list above formed fruit-bodies similar to those of the Gymnoascaceæ, but instead of asci produced rounded aleuriospore-like pycnospores borne in clusters. Several of the Gymnoascaceæ form similar bodies when grown on artificial media, apparently frequently losing the power of forming ascigerous fruits. A detailed comparison between *Trichophyton* and *Ctenomyces* shows that they possess many structures in common: method of development and arrangement of aleuriospores, clavate or fusiform swellings, etc. He concludes that the Dermatophytes should be definitely placed in the Gymnoascaceæ with a subfamily Ateleogymnoascaceæ for those genera in which ascigerous fruit-bodies have not yet been observed. He places the five species of *Trichophyton* which produce imperfect fruits in the genus *Ateleothylax*. There is thus definite evidence of the true position of these forms which has previously been assumed by some writers on unsatisfactory data.

This work leads us to the consideration of several points. All the fungi we have discussed and, in fact, the majority of fungi pathogenic to man, are known only from their imperfect stage. We know from the life-histories of fungi in general that there are many degrees in parasitism. Some fungi are restricted in their range of hosts, sometimes even having specialized strains restricted to definite hosts; further, this occurs in those rusts, known as heteroecious, which carry out part of their life-history on one host and complete it on an alternate host. On the other hand we have some forms which can live as either parasites or saprophytes. In considering the pathogenic fungi of medicine, the phenomena already recorded from cultures of other fungi—such as influence of media and various factors, the multiplicity of strains, saltations and so on—should be borne in mind. From Nannizzi's researches it appears probable that Dermatophytes were originally saprophytic on organic matter and it is possible that they may still normally occur there. We know that the genus *Actinomyces* also has a suggestive distribution—the reputed non-pathogenic forms are found principally in the soil; *Aspergillus fumigatus* occurs on seeds and in the soil, *Sporotrichum* is often saprophytic, and so on.

Certain Dermatophytes have been recorded only from particular regions, but facts upon which to base generalizations are lacking. Fungi are, however, like other organisms in their distribution, some being practically cosmopolitan, others endemic to small areas. One of the factors limiting distribution is temperature. The spores of some fungi withstand the cold of liquid hydrogen, and some fungi, as for example the dry rot fungus (*Merulius lacrymans*), grow at the temperature of frozen meat chambers. On the other hand we have species growing at the high temperatures of fermenting hay. Each species has its range of temperature; for example, *Merulius lacrymans* will not grow above 80° F., and consequently is not found in the tropics. It is necessary to study each species separately to obtain data of any value. To summarize my remarks from a systematist's point of view: it is necessary to lay more stress than usually has been done on the investigation of the fungi themselves, and this investigation, in its turn, may bring to light many important facts in the epidemiology of the mycoses.

Dr. Arthur Whitfield.

To those practising exclusively in this country the tropical mycoses have—apart from their purely scientific interest—a practical interest only in so far as they may be brought over here and become endemic.

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So far as I know, only one tropical mycosis, "dhobie itch," has been established in this country so thoroughly as to become a real menace to the health and comfort of the people.

Though fairly well recognized since Hebra's time, when it was called "eczema marginatum," only during the last thirty-five years has it been known as affecting the hands and feet.

I regard Djellaludin Moukhtar as the real discoverer of these sites of infection in the year 1892. Sabouraud says that his cases (which Sabouraud himself apparently saw) were an entirely different affection, ringworm it is true, but not belonging to the "dhobie itch" variety. Nevertheless, in the brief notices of his cases, shown before the French Dermatological Society, the fact is evident that some of the patients had the eruption not only upon the sole but between the toes. Moukhtar's error was that he really missed the importance of his own discovery by describing the condition as eczema which had become contaminated by the ringworm fungus, instead of realizing that a ringworm fungus could produce an eruption closely simulating that of non-parasitic eczema. My own contribution to this subject was published in the *Lancet* in 1908, two years before Sabouraud's article, and relates to the classical affection as we now know it. I did not, in my first few cases, succeed in cultivating the parasite, and I therefore did not attribute it to the variety epidermophyton. I did, however, lay stress on the fact that cases of so-called "gouty" and vesicular eczema of the toes were often really due to ringworm infection, and later I coined the term "eczematoid ringworm" to express the similarity of this disease to simple vesicular eczema. I also entered a strong plea for the systematic microscopic examination of material from cases of so-called vesicular eczema of the hands and feet, in order to separate the mycotic from the non-mycotic dermatoses of those regions. I was unaware of Moukhtar's observations when I published my first paper, and in excuse I may therefore point out that his consisted merely of a few cases reported in the transactions of the French society and were never, I believe, collected into a paper.

Sabouraud in 1910 wrote a long article in which he showed that the particular variety met with in his cases was the *Epidermophyton inguinale*, and he went, I think, too far in limiting the variety of fungus found in eczematoid ringworm to the *Epidermophyton* alone.

I have had cases—and others have also published reports of cases—in which the clinical picture of eczematoid ringworm has proved on culture to be due to other varieties of fungus.

Of the varieties that I have myself cultivated, by far the larger number have been the ordinary *Epidermophyton inguinale*, but I have once found *Epidermophyton niveum*, which Sabouraud says is fairly common in Paris, and I have also cultivated both an *Endothrix* and an *Ectothrix* fungus, though never a *Microsporum*.

I have several times cultivated from the interdigital horny layer and from the depth of the nail plate a penicillium fungus. One case was that of a woman, who had been resident for many years in India, and had acquired onychomycosis there. In this respect I feel that I have been very shortsighted, for I rejected all my early cultures as mere accidental contaminations. Later, it occurred to me that penicillium was not a usual contamination of skin cultures, and also that in those cases in which I had found it by culture, the immediate potash specimen, both of the nails and the horny layer, had shown fine filaments ending in a fan-shaped arrangement of spores not closely resembling *Epidermophyton*. I now believe that the fungus was *Penicillium brevicaule*, and I am on the look-out for a further case to submit to a competent mycologist. Castellani in his book refers to two cases of onychomycosis, published by Brumpt and Langeron in 1910, and found to be due to

T. brevicaule. In my cases the clinical appearances, both in the surface horny layer and in the nails, were indistinguishable from those caused by the *Epidermophyton* and other ringworm fungi.

I may also mention two other cases of onychomycosis acquired, not in the tropics but in Japan. In each of these the disease began immediately after the running of the point of a bamboo shoot beneath the nail. In one I cultivated an ectothrix fungus, but I failed to obtain it in a pure state, and in the other I failed to cultivate the fungus at all. The interest to me lies in the suggestion of an extra-animal existence of these parasitic fungi.

The last case to which I shall refer is that of a former fellow student of mine, who, while serving as medical officer with the P. and O. Line, bathed in stagnant water when in India. About four days afterwards he was attacked by an acute eczematoid inflammation of the left auditory meatus and the contiguous ear and cheek. This subsided, but relapsed again and again after he had left the sea and settled in practice in this country. During one of his attacks I saw him and microscopically discovered a fungus which proved on cultivation to be an aspergillus. Knowing little about this fungus I cannot say to which particular variety it belonged. It is perhaps noteworthy that old residents in India warned him *after* he had acquired the disease that one was apt to pick up eczema of the ear by bathing in certain places.

Dr. P. H. Manson-Bahr.

Although I have decided views on the clinical aspects and treatment of tropical skin diseases, yet from what little knowledge I possess of scientific mycology I look askance at the system of classification adopted by some authorities, at the rapidly changing nomenclature, and at the great tendency to what I must term "pecies mongering" of fungi on inadequate grounds, and in this matter I am insentire agreement with Mr. Ramsbottom.

On account of the less civilized habits of native races, the absence of adequate clothing, the heat and moisture, mycotic diseases of the skin are more in evidence in the tropics than they are in temperate climates. The first, and one of the most important fungus diseases, is mycetoma, sometimes known as *madura foot*. Although mycetoma is a disease of warm climates, it has great resemblance to actinomycosis. Originally believed to be confined to India, it is now known to have a much wider distribution, and to be found sparingly in Central Africa and South America.

The clinical appearances of the painless swelling and purulent blood-stained discharge, and the multiple sinuses, are sufficiently diagnostic. A characteristic feature about the discharge is the presence of greyish or yellowish grains usually similar to those found in actinomycosis. As regards the classification of the causal fungus we are by no means so clear. This easily recognizable clinical condition is said to be caused by at least two species of *Discomyces*, *D. bovis*, which is identical with bovine actinomycosis, and *D. maduræ*, which forms grains of a yellowish-white colour, and which produces a peculiar mode of growth by means of radiating filaments. Added to these, there are at least six forms of hyphomycetes in which reproduction takes place by conidia. Three are said to be a fungus allied to the *Aspergillus*, and a similar number to *Indiella* (*I. mansoni*, *I. regnieri*, and *I. somaliense*) have been described. But whether due to *Discomyces*, *Aspergillus* or *Indiella*, the pathology, and certainly the treatment, appear to be the same. As in actinomycosis, potassium iodide is undoubtedly of value in the early stages of the disease, but later the only chance of alleviating the condition lies in amputation.

Next I must refer to a condition known euphoniously as "mossy foot," a papillomatous condition of the feet and legs found occasionally in the Amazons,

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Honduras, and other parts of South America. The foot and ankles are covered with dense warty-looking outgrowths resembling minute barnacles. The condition has been aptly compared to dry moss growing on rocks. The sole of the foot usually escapes, but the disease is infective, and auto-inoculation from one limb to another may apparently take place.

The disease is said to be due to an infection with a hyphomycete, *Phalophora verrucosa*, described by Thaxter in 1915. It can be grown on ordinary media, in which it forms chlamydospores 8-15 μ in diameter, as well as conidia, and sometimes aerial hyphæ.

The term "blastomycosis" is given to cutaneous infection with saccharomyces or yeasts. Normally the fungi are thought to be saprophytic or live as parasites on animals or plants. They gain access to human tissues through some wound or breach of surface.

The cutaneous manifestations of blastomycosis closely resemble the verrucose type of lupus vulgaris. They appear as papules, or small nodules, of a reddish-brown colour, which gradually spread, and after months or years become irregular patches several inches in diameter.

As a rule the condition remains local, and is not associated with any disturbance of health, but in rare instances the fungi pass into the general circulation, and a septicæmic infection is the result. In the latter condition multiple abscesses form either superficially or in the internal organs. Microscopically, the lesions of cutaneous blastomycosis resemble those of lupus verrucosus.

That blastomycosis is due to a pathogenic yeast there can be little doubt, but there is considerable doubt about their classification. Two main types of fungus are recognized, a *blastomycetes* in which reproduction takes place by budding, and a *cryptococcus* in which reproduction takes place by a process of endosporulation. The former is known as *Blastomyces dermatitis* (Gilchrist and Stokes, 1898), and can be grown anaërobically, but inoculation into animals has so far proved negative. The cryptococcus forms resemble *C. farciminosus*, the cause of epizootic lymphangitis in horses. There is some doubt whether it is not identical. It sometimes produces a pulmonary disease in man, and has been described in the epithelial cells of the lung by Darling in Panama.

Almost in the same category we can classify sporotrichosis—gummatous-like swellings of the skin which break down and leave deep ulcers. The organism, *Sporotrichon beurmanni*, can be isolated and grown on Sabouraud's medium. This appears to be a common parasite in nature, being found on vegetables. In the pus, individual parasites are engulfed by the phagocytes.

The treatment of all these forms of blastomycosis is in a very unsatisfactory state, surgical measures being useless; recourse has to be had to large doses of potassium iodide. Individual tumours or lesions can be punctured and injected with potassium iodide 1 : 100. X-rays are useful as an adjunct.

Dhobie Itch.—Amongst the more cutaneous afflictions of the tropics, dhobie itch has the claim to prior place. The name is derived from the belief that it is contracted from clothes contaminated by the washerman. The disease is probably due initially to a trichophyton, *Epidermophyton inguinale*, and to the microsporon of erythrasma, *Microsporon minutissimum*. The former is not easy to grow, and does so but slowly on Sabouraud's medium.

Dhobie itch produces lesions which are very characteristic, and begin as raised, rounded, elevated papules, which spread peripherally, producing a festooned border covered with thick scales. The axilla may be affected as well as the crutch. The irritation produced may be excessive. The lesions very often disappear in the cold weather, only to reappear when the climate becomes moist and damp, and sweating is

very severe. The mycelial elements of the fungus can be recognized in scales of skin soaked in liquor potassiæ. The treatment is in quite a satisfactory state. True dhobie itch resolves, temporarily at any rate, in a striking manner to Vleminck's solution (sulphide of calcium). The solution must be freshly brewed from quicklime and precipitated sulphur. The parts should be cleansed with soap and water first, and the lotion applied for three consecutive nights on lint, held in position by bathing drawers. When this solution fails, recourse must be had to chrysophanic acid. Recrudescences are very common, even when the condition appears to have cleared up.

Pityriasis versicolor is very common in the tropics. It is the usual cause of pale or fawn-coloured scurfy patches seen on dark-skinned natives. On these dark-skinned races, contrary to what obtains in Europeans, the patch is of lighter colour than the surrounding healthy integument. The disease is usually seen in young adults and is favoured by excessive sweating. It is easily arrested by strong iodine solution or an ointment containing 3 per cent. sulphur and salicylic acid.

The desire to make tropical dermatology as complicated as possible has led certain writers to introduce the terms "tinea alba," "tinea flava" and "tinea nigra" to patches of versicolor of different hues. There is no need for any such artificial differentiation. Sir Patrick Manson, an acute and sometimes whimsical observer, while in Ceylon once caused considerable astonishment when, on being demonstrated a case of tinea nigra on a Singalese, he called for a piece of soap, a nail-brush and a towel, and converted the case in a surprisingly short space of time into a tinea alba.

Tinea imbricata or "Tokelau Ringworm" is the most characteristic of all human tropical skin diseases, and thus has a special claim to the student of tropical medicine. It was the first and certainly the most striking of skin diseases to be differentiated by Manson in 1878 in Amoy. He not only at that time recognized and drew the causative fungus, but reproduced the disease by inoculation of scales and successfully differentiated it from tinea circinata. The limpet-like concentric arrangement of the scales is quite characteristic.

The disease usually occupies a large area on the body, it does not affect the palms and soles or the scalp. The scales are like tissue-paper and, when they are removed the mycelium of the fungus may be easily recognized under the microscope. Two species of fungi, *Endodermophyton concentricum* and *E. indicum*, have been recognized as the cause of the disease and have been grown by immersing the scales in alcohol for five to ten minutes and then placing one scale to each tube of glucose broth. It is difficult to believe that such a characteristic disease is due to two separate species of fungi.

The treatment in the early stages is by painting with strong iodine; or by salicylic acid (gr. xv to the oz.) or by chrysophanic acid. Since the practice of oiling the skin has been in evidence in the Pacific Islands, tinea imbricata has to a great extent disappeared.

Pinta or *Caraate*.—This most remarkable pigmentative skin disease is limited to the New World. It probably was a disease peculiar to the Aztecs and Inca and was noted by the Conquisatores of Peru. The disease starts at one or two points, the rest of the skin becoming infected in turn by extension or auto-contagion. At first, hands, face and other exposed parts are attacked. The original patch becomes hyperpigmented owing to active pigment-formation but later becomes white, red, blue or black. As the patches spread they assume a variety of shapes.

If the scales are moistened with liquor potassiæ the black spores and highly refractile mycelium are found. These spores are round or oval 8 to 12 μ. Montoya, in his well-known thesis in 1898, described as many as twenty varieties of the fungus, but we have been spared one thing: he never christened them with specific names! To him, then, we owe a debt of gratitude. Apparently the fungus has never

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been cultivated, though the disease has been transmitted by inoculation. Treatment is said to be easy and the patches rapidly yield to chrysophanic acid and a liniment of iodine and preparations of sulphur.

Several other well-defined mycotic conditions must be referred to. There is "Hong Kong foot," a mycotic infection of the soles and the intradigital spaces, occurring in China, especially during the summer months. It appears as deep-seated vesicles on the inner margin and hollow of the sole and as a macerated condition of the skin of the intradigital clefts; a persistent itching is a marked feature. Dold has described a fungus as resembling *E. inguinale* but it has not yet received the study it deserves. This intractable and distressing condition is contracted, it is believed, through wearing improperly tanned boots. It is particularly difficult to cure. Bathing in sea water has a great effect, but in advanced cases recourse must be had to X-rays. Hong-Kong foot is probably the same condition as the mycotic eczema so well described by Dr. Whitfield. *Prickly heat* was formerly thought to be due to excessive perspiration, but now, owing to the work of E. C. Smith, of Lagos, it is probable that the condition is due to an infection of the sweat glands and ducts with a small yeast or monilia. This investigator has reproduced the condition by rubbing cultures of this organism into the skin. Prickly heat is a miliary-like eruption too familiar to need description; those who have suffered from it will remember it with sympathy. Treatment is on general lines, but a soap containing oxycyanide of mercury, known as Afridol, when applied and allowed to dry, appears to act in a preventive as well as a curative manner.

The clinical aspects of tropical skin diseases are, for the most part, well-defined; but much remains to be done in isolation and identification of the fungi.

The greatest medical mycologist is he who would classify these organisms on a true botanical basis and help to stabilize the nomenclature on true scientific lines.

Vice-Admiral A. Gaskell, C.B. (War Section)

said that naval people were interested in the diseases which had been mentioned, especially in what naval men called "toe rot," and in "dhobie itch," which were recognized clinically by naval practitioners who knew what havoc they caused, not only among dirty patients but also among the clean type to which the men in the Navy belonged.

Dr. G. B. Dowling

said that for some years Mr. W. G. Garner, the pathological assistant at St. John's Hospital, had made cultures from the pityrosporon of Sabouraud, or the so-called "bottle bacillus" or "spore of Malassez." Dr. MacLeod had made a mycological study of the organism, and he (the speaker) had been engaged in clinical investigations on its behaviour when implanted on human skin. He had taken a number of cases of seborrhœic dermatitis, and treated them with living cultures of this organism, in two ways. The organism had been planted on the skin after light scarification and it had been inoculated intradermally in a form of suspension in water. After scarification the organism had been taken straight from culture, dry, and put on, and the result was watched. It had been found that a patch of seborrhœic dermatitis could always be produced by both those methods, though more easily by scarification. The disease, artificially produced in this way, tended to imitate the type of lesion that the patient already had. On normals and on those already suffering from seborrhœic dermatitis, the earliest reaction to the organism was a group of follicular papules.

[Some photographs of these artificially produced symptoms were shown.]

The conclusions formed from this investigation were: (1) that seborrhœic dermatitis was an infection due solely to the spore of Malassez; (2) that the organism flourished on moist and seborrhœic skins.

Mr. Ramsbottom (in reply)

said, with regard to Dr. Manson-Bahr's remarks concerning systematists, that when organisms were obviously different, it was well to assign to them different names. Certain points he had at times brought before meetings of the Section of Tropical Diseases would perhaps rejoice Dr. Manson-Bahr's heart. There were, among the cultures shown by Dr. Broughton-Alcock, four apparently different fungi isolated from one case, and he (the speaker) thought the probability of obtaining four different pathogenic fungi was rather outside one's ideas of chance. However, to give a name to what was found, without describing it properly, was very bad science.

Dr. A. Whitfield (in reply)

said that "Hong Kong foot" was obviously eczematoid ringworm; he regarded it as probably the same condition as "mango toe," and certainly the same as "lascar foot." Passengers on board ship in the tropics shaved, then walked on deck in pyjamas with bare feet, where a lascar had already gone with bare feet. They thus picked up the disease and carried it to the stateroom, and the next inhabitant of the stateroom became infected too. It was not an easy condition to treat; dhobie itch in the groin showed a great tendency to relapse. He used an ointment for it, consisting of 25 gr. benzoic acid, 15 gr. salicylic acid, 2 drachms of vaseline, and coconut-oil to the ounce. This would cure ringworm in the groin, and in a number of cases on the foot, without pain and with little disturbance or staining. In the foot, the early attack was frequently bullous, and it was during the acute attack that cure should be attempted. It might be mistaken for an acute vesicular eczema of the foot, but if it were soothed with lead lotion it became chronic, and thereafter was very resistant. The ointment mentioned caused the patient to shed his skin in the same way as the canary shed its feathers. He instructed patients to wear boiled cotton socks continually, and to destroy old bedroom slippers. During the last year or two he had modified his benzoic acid treatment into a very clean and efficacious method. He used the same strength, but dissolved it in one part of acetone to three parts of spirit. The great need was to keep the foot dry. Acetone spirit was very desiccating, whereas ointments macerated.

Dr. Manson-Bahr (in reply)

said that he had had no intention of criticizing Mr. Ramsbottom. It was a great advantage to obtain the views of a professed mycologist. His (the speaker's) contention was that the beginning was made from the wrong end. The true botanist should be consulted before names were applied; when the reverse course was pursued it only resulted in confusion, especially to students.

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LES EPIDERMOMYCOSES.

Par le Dr. MAURICE LANGERON.

(Chef de Laboratoire à la Faculté de Médecine de Paris.)

COMMUNICATED BY REQUEST.

SOUS le nom d'épidermomycoses, on peut réunir toutes les dermatoses localisées à l'épiderme et attribuées à des champignons. On doit donc exclure des épidermomycoses les mycoses cutanées, qui atteignent aussi le derme et qui produisent des végétations ou des ulcérations plus ou moins profondes, telles que celles qu'on observe dans les dermatites blastomycosiques, les dermatites verruqueuses, etc. Il faut en exclure aussi les maladies mycosiques des poils ou trichomycoses d'origines diverses (teignes, trichospories, maladies nodulaires, etc.) et les onychomycoses ou maladies mycosiques des ongles. Ces dernières présentent pourtant, avec les épidermomycoses, ce point commun de pouvoir être produites par des champignons très variés. On y a trouvé en effet un saccharomycète ascosporé, le *Debaryomyces gruetzi* Ota, 1924, des Clostérosporés (*Trichophyton felineum* R. Blanchard, 1895, et *Grubyella schönleini* Lebert, 1845), des Ascomycètes périsporiacés appartenant aux genres *Aspergillus* (*A. jeanselmei* Ota, 1923, et *A. gratioti* Sartory, 1920), *Sterigmatocystis* (*S. unguis* Weil et Gaudin, 1919) et *Scopulariopsis* (*S. brevicaulis* var. *hominis* Brumpt et Langeron, 1910; *S. cinerea* Weil et Gaudin, 1919), et même le *Rhinocladium beurmanni*.

Au point de vue étiologique, on peut diviser les épidermomycoses en deux grands groupes. Les unes sont en quelque sorte *spécifiques* et produites par des champignons nettement pathogènes, vivant dans les squames épidermiques, cultivables et inoculables. Le type clinique est généralement bien accentué: herpès circiné, eczéma marginé, pityriasis, achromie ou hyperchromie, et permet de diagnostiquer d'emblée l'origine mycosique, quelquefois même le groupe de champignons auquel appartient le parasite. A cette catégorie on peut rattacher toutes les épidermomycoses à dermatophytes clostérosporés (épidermophyties, endodermophyties, épidermomycoses trichophytiques et faviformes), à dématiées (*Cladosporium* or *Hormodendron*), pityriasiques et enfin l'erythrasma.

Les autres sont extrêmement polymorphes au point de vue clinique. Elles constituent les épidermomycoses proprement dites, sur lesquelles l'attention des dermatologistes n'est attirée que depuis peu d'années. Le premier travail d'ensemble sur cette question est dû à Petges (de Bordeaux) et a été présenté au 1er Congrès des dermatologistes et syphiligraphes de langue français, tenu à Paris du 6 au 8 juin 1922. Parmi les travaux français, il importe de signaler aussi diverses publications de Dubreuilh, Dubreuilh et Joulia, sur les intertrigos mycosiques, la thèse de Schoux (1920) sur l'épidermophytie inguinale, celle de Touraine-Desvaux (1923), sur les épidermomycoses à levures et à champignons levuriformes. Les travaux de Lombardo, Mitchell, Ota, White et Greenwood sont de la même époque. Tout récemment (1926) l'enquête de Keller, faite à Fribourg-en-Brisgau, et celle de Falchi, faite à Pavie, fournissent des statistiques très intéressantes.

Keller, sur 409 cas, trouve 194 mycoses, dont 138 ont donné des cultures positives. Sur ces 138 cas, il y avait 9 microspories cutanées, 2 épidermomycoses faviformes à *Sabouraudites gypseus* (Bodin, 1907), 6 épidermophyties inguinales, dont 4 à *Sabouraudites ruber* (Castellani, 1909) et 2 à *Sabouraudites lanoroseus* (MacCarthy, 1925), 27 dyshidroses des mains et des pieds et 57 trichophyties, parmi lesquelles 10 étaient localisées aux mains et dues à *Sabouraudites persicolor* (Sabouraud, 1910), 17 étaient des trichophyties herpétiformes à *Sabouraudites quinckeanus* (Zopf, 1890); il y avait enfin 10 cas d'épidermites à blastosporés et 2

cas de sporotrichose à *Rhinocladium beurmanni* (Matruchot et Ramon, 1905). Les dyshidroses ont été étudiées avec un soin particulier. Le champignon de Kaufman-Wolf a paru localisé surtout aux orteils et à la plante des pieds, tandis que les dyshidroses des doigts étaient dues exclusivement à des blastosporés. L'opinion de Keller sur le champignon de Kaufman-Wolf est très voisine de celle à laquelle Ota et moi-même sommes arrivés : on peut le considérer comme un *Trichophyton* très voisin du *Sabouraudites interdigitalis* (Priestley, 1917), dont il est probablement une forme pléomorphique naturelle ; c'est pourquoi nous l'avons rangé dans notre genre *Trichophyton sensu stricto*. Les catégories de dyshidroses adoptées par Keller sont à peu près les mêmes que celles que nous venons d'exposer. Ce sont : 1° les érosions digitales à blastosporés ; 2° les épidermophyties dyshidrosiformes proprement dites ; 3° les trichophyties des extrémités. Keller distingue encore deux groupes de dyshidroses à forme vésiculeuse, dans lesquelles il n'a pu déceler de champignons et dont les vésicules sont les unes à contenu alcalin, les autres à contenu acide.

Tandis que Keller s'est surtout attaché à l'étude des dermatophytes clostérosporés, G. Falchi accorde une grande importance aux épidermomycoses à blastosporés. Il leur attribue la dermatite érythémato-squameuse des enfants, les mycoses interdigitales, la plupart des lésions intertrigineuses, beaucoup d'onychomycoses, et fait remarquer que, malgré la résistance des dermatologistes, il faudra bien admettre tôt ou tard le rôle pathogène de ces champignons. Falchi démontre que, sur la peau saine, on trouve surtout des périsporiacées et des mucoracées, bien plus rarement des blastosporés. Il est donc difficile d'admettre que la présence des blastosporés trouvés dans les épidermomycoses soit un fait banal et fortuit : leur rôle pathogène est évidemment difficile à démontrer, mais ce ne sont pas des hôtes habituels de la peau.

Dans les épidermomycoses polymorphes, les caractères morphologiques cliniques ne permettent pas d'établir ni même de présumer la nature parasitaire de l'affection ; les épidermomycoses parasitaires de ce groupe ne se distinguent en rien des dermatoses analogues non parasitaires. En outre il n'y a pas de spécificité : les lésions sont produites comme nous venons de le voir par des champignons très variés ; dermatophytes proprement dits ou Clostérosporés, Blastosporés ascosporés ou anascosporés, Aleuriosporés, Microsiphonés, etc. Dans ce groupe polymorphe, les dermatologistes, suivant leurs tendances plus ou moins analytiques, ont distingué de nombreux types d'épidermomycoses. Ceux-ci, pour la clarté de l'exposé, peuvent se ramener à quatre types principaux. Ce sont : le type intertrigineux, le type vésiculeux ou dyshidrosiforme, le type maculeux ou eczématiforme et le type pityriasique.

(1) Epidermomycoses spécifiques.

La classification de ces épidermomycoses, dont l'agent est bien connu, constant et généralement facile à isoler, doit être une classification étiologique ou parasitologique, car ici les signes cliniques passent au second plan et peuvent être communs à plusieurs épidermomycoses produites par des champignons différents.

(1°) *Epidermophyties proprement dites.*—Le type de ces épidermophyties est l'eczéma marginatum de Hebra. Cette dermatose est produite par des champignons appartenant au genre *Epidermophyton* Lang, 1879, tel qu'il est compris par Ota et Langeron, c'est-à-dire caractérisé par la rareté des aleuries et par l'abondance des fuseaux. Provisoirement, ce genre est réduit à deux espèces : *Epidermophyton cruris* (Castellani, 1905) = *E. inguinale* (Sabouraud, 1907) et *E. clypeiforme* (MacCarthy, 1925). Les autres espèces sont trop insuffisamment caractérisées pour qu'on puisse établir avec précision leur position systématique. Ceci correspond d'ailleurs au groupe des *Epidermophyton* monomorphes de MacCarthy. Si ce dermatologiste

n'attribuait pas un sens clinique au mot *epidermophyton* et s'il raisonnait en botaniste systématicien, il serait exactement d'accord avec nous et ferait de ses autres espèces, formant, outre les fuseaux, des grappes et des thyrses d'aleuries, des *Sabouraudites* du sous-genre *Aleurocloster*.

(2°) *Endodermophyties.*—Ces dermatoses, d'aspect clinique très spécial, sont dues à des dermatophytes particuliers, formant le genre *Endodermophyton*, dont on doit la connaissance à Castellani (1909). Ces champignons diffèrent profondément des *Epidermophyton* par leur morphologie très pauvre et sont voisins, à ce point de vue, du genre *Bodinia* Ota et Langeron, 1923 : ils ne forment guère que des arthrospores en chaînettes, il n'y a pas de fuseaux et les aleuries sont rares et rudimentaires. Les arthrospores des *Endodermophyton* se désarticulent encore plus difficilement que celles des *Bodinia*. Trois espèces d'*Endodermophyton* produisent les dermatoses en cocardes concentriques, connues sous le nom de tokelau ou de tinea imbricata. Ce sont : *E. concentricum* (R. Blanchard, 1895), espèce type du genre, *E. indicum* (Castellani, 1911) et *E. mansoni* (Castellani, 1914). En outre, da Fonseca a isolé au Brésil l'*E. roquettei* (da Fonseca, 1925) d'une autre dermatose, le chimbéré, sorte de caraté fréquent chez les Indiens du fleuve San Miguel au Matto Grosso (Brésil).

(3°) *Epidermomycoses faviformes.*—Le favus de la peau glabre est une simple épidermomycose lorsqu'il prend la forme épidermique circinée, sans godets, ou favus herpeticus. On sait que ces lésions sont plus régulières que celles de l'herpès circiné trichophytique. On en a isolé plusieurs espèces : *Grubyella schönleini* (Lebert, 1845), agent des lésions faviques classiques, et *G. alba* (Sabouraud, 1909), qui produit uniquement une épidermomycose. Ce champignon, observé d'abord en France par Sabouraud, a été fréquemment retrouvé par P. Horta au Brésil.

(4°) *Epidermomycoses trichophytiques ou Trichophyties cutanées.*—Les trichophyties cutanées prennent généralement la forme classique de l'herpès circiné, qui peut être plus ou moins bien caractérisé cliniquement. De très nombreuses espèces ont été trouvées dans ces lésions. Ce sont : *Trichophyton coccineum* Kato, 1926, espèce japonaise qui produit aussi de la dyshidrose ; *T. depressum* MacCarthy, 1925, *T. sabouraudi* R. Blanchard, 1895, *T. sulphureum* Fox, 1908, *T. tonsurans* (Malmsten, 1845), *Sabouraudites audouini* (Gruby, 1843), *S. felineus* (Fox et Blaxall, 1896), *S. flavescens* (P. Horta, 1911), espèce brésilienne ; *S. gallinæ* (Mégnin, 1881) (=*Achorion gallinæ* Sabouraud, 1910), champignon qui donne des lésions exclusivement superficielles, sans rapport avec le favus, aussi bien chez l'homme que chez la poule, comme l'a montré Altara (1925) ; *S. griseus* (Vasconcellos, 1914), épidermophyte brésilienne ; *S. gypseus* (Bodin, 1907) (=*Achorion gypseum* Bodin, 1907), isolé d'un favus d'apparence clinique classique ; *S. japonicus* (Dahi et Kambayashi) très fréquent à Formose (42 cas sur 92) ; *S. lanoroseus* (MacCarthy, 1925), et *S. plurizoniformis* (MacCarthy, 1925), tous deux exclusivement épidermophytes ; *S. lanosus* (Sabouraud, 1907) (=*Microsporum lanosum* Sabouraud, 1907), *S. scorteus* (Priestley, 1914) isolé au Queensland tropical d'une épidermophytie cutanée infantile microsporique et probablement identique à *S. fulvus* (Uriburu, 1907) trouvé par Uriburu en Argentine ; *Bodinia violacea* (Bodin, 1902), *B. spadix* Kato, 1926, connu seulement au Japon.

On a encore signalé, dans des épidermophyties, divers dermatophytes dont la position systématique est encore incertaine à cause de l'insuffisance des descriptions. Ce sont : *Trickophyton castellanii* Perry, 1918, trouvé à Ceylan par Castellani dans une dermatose spéciale (tinea intersecta), caractérisée par des papules craquelées ; *T. ceylonense* Castellani, 1908, isolé par Castellani à Ceylan d'une dermatose qu'il nomme tinea nigro circinata ; *T. macfadyeni* Castellani, 1905.

(5°) *Epidermomycoses achromiques ou hyperchromiques.*—On peut réunir sous ce titre les épidermomycoses dans lesquelles un champignon spécifique produit une

coloration particulière ou au contraire une décoloration de l'épiderme. Le pityriasis versicolor est caractérisé par des taches bien limitées, dont la coloration peut varier du jaune pâle au brun foncé ; quelquefois il s'y mêle une teinte rosée. Cette épidermomycose est produite par le *Malassezia furfur* (Ch. Robin, 1852), toujours très abondant dans les squames, mais très difficile à cultiver. Deux épidermomycoses très voisines ont été décrites à Ceylan par Castellani : ce sont le pityriasis versicolor flava ou tinea rosea, produit par le *Malassezia tropica* Castellani, 1908, et la tinea alba, produite par le *Malassezia macfadyeni* Castellani, 1918. Une autre épidermomycose de même nature et plus anciennement connue est la tinea nigra, découverte par Manson dans le sud de la Chine. Elle paraît commune à Ceylan, à Java, en Birmanie et en Malaisie. Elle est produite par une dématiée, le *Cladosporium mansoni* (Castellani, 1908), champignon donnant en culture des colonies très foncées, couvertes de conidies extrêmement caduques, possédant des facettes d'insertion avec disjoncteurs. Une espèce très voisine, *C. wernecki*, Horta, 1921, a été isolée au Brésil par P. Horta d'une épidermomycose ressemblant beaucoup à la tinea nigra ; j'ai pu faire avec Horta l'étude de ce champignon et décrire, outre la forme *Cladosporium*, des formes blastosporées des types *Dematium* et *Fumago*.

Jeanselme en Indo-Chine, Fontoynont et Carougeau à Madagascar ont observé une épidermomycose caractérisée par des plaques achromiques quelquefois saisonnières et par de très nombreux filaments mycéliens dans les squames épidermiques. Fontoynont et Carougeau ont cultivé ce champignon que j'ai décrit sous le nom d'*Hormodendron fontoynonti* Langeron, 1913. C'est certainement l'agent de cette épidermomycose achromique, car, en partant des cultures, Fontoynont et Carougeau ont pu reproduire expérimentalment la lésion sur l'homme. Les *Hormodendron* sont très voisins des *Cladosporium* et s'en distinguent facilement par la présence d'un conidiophore nettement différencié, portant des chaînettes ramifées de conidies très caduques.

Jeanselme a signalé pour la première fois en Indo-Chine une autre achromie parasitaire, connue des indigènes sous le nom de khihuen ; c'est ce que les Siamois nomment khi et les dermatologistes tinea albigena. Cette épidermomycose est produite par un aleuriosporé, le *Glenospora albiciscans* (Nieuwenhuis, 1907). Ce champignon avait d'abord été considéré comme un *Trichophyton*, mais Ota a démontré que c'est un aleuriosporé, c'est-à-dire un champignon dont les spores imparfaites ou aleuries ne se détachent pas du mycélium, et l'a placé dans le genre *Glenospora*.

On peut enfin rattacher à ce groupe d'épidermomycoses l'érythrasma, dont les plaques polycycliques, de couleur plus ou moins rouge ou brunâtre ou même bistrées, sont dues à un microsiphoné, l'*Actinomyces minutissimus* (Burchardt, 1859). La position systématique de ce champignon, découvert par Burchardt et bien décrit par Balzer, est restée longtemps incertaine. On le rattache maintenant aux microsiphonés ou actinomycètes dont il présente les caractères morphologiques.

(II) Epidermomycoses polymorphes non spécifiques,

Pour cette catégorie d'épidermomycoses, il est nécessaire d'adopter une classification clinique, parce que les agents pathogènes sont très variés et multiples pour chaque type de dermatose. Cette classification est bien moins précise que la classification étiologique parce que souvent les types cliniques ne sont pas très tranchés et passent de l'un à l'autre par des transitions insensibles.

(1°) *Epidermomycoses intertrigineuses.*— Le diagnostic étiologique de ces lésions ne peut être fait que par les méthodes de laboratoire ; il semble bien qu'aucun signe clinique ne permet de décider si l'intertrigo est dû à un streptocoque, à un dermatophyte ou à un champignon levuriforme. Quel que soit l'agent pathogène, la surface

des plis cutanés réagit de la même façon. Toutefois, lorsqu'il s'agit d'un intertrigo produit par des blastosporés, la surface de la lésion est plus humide et recouverte, surtout au fond des plis, d'un épais enduit blanchâtre.

Très souvent l'intertrigo est dû à l'*Epidermophyton cruris* (Castellani, 1905), mais on a isolé aussi de ces lésions des blastosporés et des arthrosporés. Les seuls blastosporés étudiés avec précision sont deux levures ascosporées décrites par Ota en 1924 : *Debaryomyces fabryi* et *D. tremoniensis*, toutes deux isolées par Fabry à Dortmund, en 1917, de lésions interdigitales. Burnier et Touraine-Desvaux ont isolé à l'Hôpital St. Louis, à Paris, plusieurs souches de *Monilia* et de *Cryptococcus* : ces souches ont été étudiées soit par Ota, soit par moi-même. Il ne nous a pas été possible d'en donner une détermination spécifique, pas plus que pour le *Cryptococcus* isolé en 1914 par Gougerot et Gancea d'une épidermomycose des plis inguinaux.

Tout ce qu'il est possible de faire provisoirement pour les formes anascosporées c'est de les répartir dans les deux groupes *Monilia* et *Cryptococcus*, suivant qu'elles produisent ou non des filaments portant des verticilles de blastospores, après ensemencement dans des milieux liquides pauvres, tels que l'eau de pommes de terre. Nos connaissances ne permettent pas encore d'aller plus loin, à moins qu'on n'ait recours au procédé simpliste qui consiste à créer une espèce nouvelle pour chaque souche ; mais on serait, je crois, bien embarrassé pour les caractériser.

A ma connaissance, on n'a encore publié qu'un cas d'épidermomycose intertrigineuse produite par un arthrosporé, c'est celui dans lequel Falchi a rencontré *Mycoderma lactis* Link, 1809.

(2°) *Epidermomycoses dyshidrosiformes.*—Ces épidermomycoses ne sont bien entendu pas toutes mycosiques. La dyshidrose est un syndrome qui peut être réalisé aussi par une irritation physique ou chimique ou même sans cause appréciable. Les lésions d'origine mycosique sont limitées aux mains ou aux pieds ; elles siègent de préférence dans les espaces interdigitaux, plus rarement aux faces palmaire ou plantaire. Aux mains, le troisième espace interdigital est le plus fréquemment atteint, tandis que le premier est souvent épargné. L'élément fondamental est la vésicule, mais l'épiderme peut aussi s'exfolier en larges lambeaux et il se produit du suintement et de la macération. Ces lésions sont très persistantes et très difficiles à guérir.

Dans les épidermomycoses dyshidrosiformes on a trouvé plusieurs dermatophytes clostérosporés. C'est tout d'abord *Epidermophyton cruris* (Castellani, 1905) qui paraît être de beaucoup le parasite le plus fréquent et qui établit ainsi un lien entre les épidermomycoses dyshidrosiformes et les épidermophyties proprement dites. Vient ensuite le *Trichophyton felineum* R. Blanchard, 1895 (= *T. radians* Sabouraud, 1909, *T. niveum* Sabouraud), agent de l'herpès iris vésiculeux de Biett ou trichophytie circinée dyshidrosiforme de Sabouraud. Ota a isolé à Moukden, en Mandchourie, une espèce très voisine, le *Trichophyton pedis* Ota, 1922, qu'il a rencontré dans quatre cas de dyshidrose du pied. Enfin Priestley avait déjà fait connaître, en 1917, le *Sabouraudites interdigitalis* (Priestley, 1917) qu'il avait rencontré dans divers cas de dyshidrose des mains et des pieds.

Parmi les blastosporés, on ne connaît encore qu'un petit nombre d'espèces déterminées avec précision. Le *Cryptococcus jeanselmei* Burnier et Langeron, 1922, provient de lésions de ce groupe. Le *Schizosaccharomyces hominis* Benedek, 1927, dont il sera question à propos des épidermomycoses eczématiformes et séborréiques, ne paraît jouer qu'un rôle assez effacé dans les lésions dyshidrosiformes. Pollacci et Nannizzi ont décrit en 1926, sous le nom de *Cryptococcus interdigitalis*, un blastosporé rencontré en Italie dans des lésions dyshidrosiformes interdigitales. Ce champignon a été isolé par Lombardo à Sassari et à Pise, par Truffi à Savone, par Rebaudi et Marengo à Gênes, par Falchi à Pavie. Ce champignon est en réalité un *Monilia*.

Sous peine de retomber dans une inextricable confusion, il faut, si on conserve le genre *Cryptococcus*, n'y faire entrer que les levures anascosporées ne donnant jamais de formes filamenteuses, même en milieux liquides pauvres. Or le champignon décrit par Pollacci et Nannizzi produit des filaments chargés de verticilles de blastospores. Il présente donc les caractères établis par Vuillemin pour le genre *Monilia* et doit se nommer *Monilia interdigitalis* (Pollacci et Nannizzi, 1926). Falchi a d'ailleurs isolé d'un cas analogue un champignon qui a été déterminé comme *Monilia*. F. Silva a aussi observé à Bahia, en 1923, plusieurs cas d'intertrigo à blastosporés et en a fait une étude complète.

A Paris, à l'Hôpital St. Louis, Burnier et Touraine-Desvaux ont trouvé dans des épidermomycoses de ce type, tantôt des *Cryptococcus*, tantôt des *Monilia*. J'ai pu examiner ces cultures qui m'ont été aimablement communiquées par le Dr. Burnier. Par repiquage sur eau de pommes de terre, j'ai pu facilement séparer les champignons appartenant à ces deux groupes, les uns (*Cryptococcus*) ne donnant jamais de filaments et seulement des formes bourgeonnantes de petite taille, les autres (*Monilia*) donnant des filaments plus ou moins ramifiés et allongés, portant des verticilles de blastospores et des chlamydospores terminales, quelquefois très nombreuses, comme dans le véritable *Monilia albicans* (Ch. Robin, 1853). Ce principe a été d'ailleurs adopté par Ciferri et Redaelli dans leur récente classification des Torulopsidacées (1925) qui complète les travaux d'Ota et de Berkhout. Ciferri réunit dans la famille des Torulopsidacées toutes les levures asporogènes pouvant ou non former un mycelium. Ciferri et Redaelli la divisent en deux sous-familles : les Cryptococcacées, entièrement dépourvues de mycelium, et les Mycotorulées qui peuvent former un mycélium un plus ou moins rudimentaire. Le genre *Cryptococcus* Kützing, 1833, maintenu par Vuillemin pour les levures asporogènes parasites de l'homme et des animaux, disparaît comme mal caractérisé au point de vue botanique et trop médical. Ses représentants sont distribués dans les trois genres : *Klöckeria* Janke, 1923, *Eutorulopsis* Ciferri, 1925, et *Torulopsis* Berlese, 1894.

La place des anciens *Monilia* dans les Mycotorulées est moins bien définie et c'est là le point faible de la classification de Ciferri et Redaelli. Ces espèces se répartissent entre les deux genres *Candida* Berkhout, 1923, et *Mycotorula* Will, 1917, et les moins différenciés restent dans le genre *Blastodendrion* Ota, 1924, conservé comme intermédiare à titre provisoire.

Ota avait déjà mentionné, en 1924, quatre souches provenant de mycoses interdigitales sous les noms de *Mycelorhizodes cutaneum* (cas de Fabry à Dortmund et de Grütz à Kiel), *M. gruetzi* (cas de Grütz à Kiel) et *Monilia* sp. (cas de Kaufmann-Wolf), mais sans donner de description de ces espèces. Ces hésitations montrent à quelles difficultés on se heurte quand on veut préciser la systématique de ce groupe difficile, dont la morphologie est à la fois si pauvre et si uniforme.

(3°) *Epidermomycoses eczematiformes.*—Comme l'eczéma vrai, ces épidermomycoses peuvent être vésiculeuses, papuleuses ou même séborréiques. Les formes vésiculeuse et papuleuse ne sont pas très distinctes l'une de l'autre : les vésicules ou papules sont rapidement déchirées par suite du prurit et laissent écouler une sérosité citrine. La forme séborréique est le plus souvent sèche et squameuse. Les lésions apparaissent de préférence au voisinage des plis, mais elles peuvent se manifester aussi en des points quelconques de la surface des téguments.

On a signalé dans ces épidermomycoses des champignons très variés et même le *Rhinocladium beurmanni*. Le type le plus accentué de ces lésions eczématoïdes est l'eczéma marginatum de Hebra, produit par l'*Epidermophyton cruris* Castellani, 1905, que nous avons mentionné comme type des épidermomycoses spécifiques. D'autres dermatophytes clostérosporés ont été trouvés dans des lésions eczématiformes : *Sabouraudites ruber* (Castellani, 1909) paraît être en Extrême-Orient, notamment au Japon et en Mandchourie, le principal agent de l'eczéma

marginatum, de l'herpès circiné et des épidermites dyshidrosiformes ; Ota en signale plus de 40 cas. Ce champignon est très probablement identique au *Trichophyton purpureum* Bang, 1910. Le *Trichophyton minimum* Le Calvé et Malherbe, 1898, peut passer du poulain à l'homme et produire des lésions erythémateuses fugaces. Enfin Castellani a signalé à Ceylan, en 1912, une espèce particulière, qu'il nomme *Trichophyton nodoformans*, mais dont la description est trop sommaire pour permettre de le situer exactement parmi les Clostérosporés.

Parmi les blastosporés, il importe de signaler les espèces suivantes :

Endomyces crateriforme Hudelo, Sartory et Montlaur, 1920, champignon isolé par ces auteurs d'une épidermomycose eczématoïde de la région inguino-périnéale et des espaces interdigitaux ; à défaut d'une description et de figures suffisamment précises, la position de ce blastosporé dans le genre *Endomyces* reste douteuse.

Debaryomyces burnieri Ota, 1924, provient des souches isolées par Burnier à l'Hôpital S. Louis, à Paris, en partant d'épidermomycoses eczémateuses. Il y avait aussi parmi ces souches de nombreuses formes de *Cryptococcus* et de *Monilia*.

Schizosaccharomyces hominis Benedek, 1927, a été isolé à Leipzig dans 50 cas d'eczéma séborréique et d'épidermomycoses érythémato-squameuses, dont il semble bien qu'il soit l'agent pathogène.

Falchi a rencontré un arthrosporé, *Mycoderma lactis* Link, 1809, dans une épidermomycose de type érythémato-vésiculaire.

Une particularité remarquable est la découverte, dans la région lyonnaise, de toute une série d'aleuriosporés dans des lésions trichophytoïdes ou érythémato squameuses. Ce sont trois espèces du genre *Aleurisma* (Link, 1809), Vuillemin, 1910 : *A. arloingi* (Busquet, 1899), découvert en 1889 par Désiré de Fortunet et Courmont, *A. lugdunense* Massia et Grigoraki, 1924, et *A. guilliermondi* Grigoraki, 1927. Le *Corethropsis hominis* Vuillemin, 1913, isolé à Nancy d'une lésion trichophytoïde par Spillmann et Jannin, appartient au même groupe. Enfin, c'est à ce groupe de lésions qu'on peut rattacher les épidermomycoses produites par deux champignons découverts à Leipzig par Benedek : *Sporotrichum lipsieux* et *Cephalosporium niveolanosum*.

(4°) *Epidermomycoses pityriasiques.*—Parmi les principales formes d'épidermomycoses pityriasiques, il importe de signaler : le pityriasis simplex, produit par les *Pityrosporum* Sabouraud, 1895 : *P. ovale* (Bizzozero, 1882) et *P. cantliei* (Castellani, 1908) ; le pityriasis circinata et marginata, dans lequel on a trouvé deux microsiphonés : *Actinomyces dispar* (Vidal, 1882) et *A. decussatus* (Langeron et Chevallier, 1912), dont le rôle pathogène est d'ailleurs très douteux. En Louisiane, Castellani a rencontré, dans des placards pityriasiques, un champignon qu'il range dans le genre *Trichophyton* et qu'il nomme *T. louisianicum* Castellani, 1927.

(5°) *Epidermomycoses achromiques ou hyperchromiques.*—Ce sont les *caratés*. Ce groupe est homologue de celui que nous avons étudié dans les épidermomycoses spécifiques, mais il s'agit ici de lésions très polymorphes et probablement produites par des champignons très variés. Pourtant ce groupe est très homogène par l'ensemble des signes cliniques. Ce sont des dermatoses desquamatives et prurigineuses, formant de larges plaques ou placards à contours géographiques, souvent très étendus. Ces placards ont pour caractéristique constante d'être achromiques ou colorés et présentent alors des teintes très variées, allant du gris au noir, ou encore bleues, violettes, rouges, etc.

Il y a des caratés achromiques, comme le chimbéré du Brésil, que nous avons signalé parmi les dermatoses spécifiques. Le kourou-kourou est très répandu le long des affluents de l'Amazone : des tribus indiennes en sont atteintes en totalité. Les caratés de Colombie atteignent, d'après Montoya y Florez, 4 pour cent de la population : le caraté violet s'attaque surtout aux paysans et aux mineurs, les caratés noir et blanc à la race noire, le caraté rouge aux citadins, les variétés bleuâtre et rougeâtre

aux blancs qui vivent à la campagne. Il est probable que la pinta ou mal del pinto du Mexique et du Yucatan, le cute du Venezuela, le quirica et autres dermatoses colorées américaines rentrent dans cette catégorie d'épidermomycoses, mais ces affections sont encore à peine étudiées. Les travaux de Montoya y Florez, faits en 1893 avec les techniques imparfaites de cette époque, sont à reprendre; il est fort probable que les *Penicillium* et *Aspergillus* trouvés alors dans les squames sont des saprophytes banaux, développés sur les parties mortes de l'épiderme. C'est ce qui est arrivé pour le tokelau, dans lequel Tribondeau avait cru voir un *Aspergillus*. Pourtant Spegazzini aurait trouvé, en Argentine, dans une dermatose comparable au caraté blanc, un *Aspergillus* (*A. dessyi*) du groupe *fumigatus*. Castellani (1925) a rapproché certains caratés jaunes de la tinea flava à *Malassezia tropica*, le caraté noir de la tinea nigra à *Cladosporium mansoni;* il a pensée que d'autres caratés, blancs, noirs, bleus ou bronzes, peuvent être consécutifs à des trichophyties cutanées. Notons enfin que le *Trichophyton carateum* Brumpt, 1913, a été découvert par Darier dans les squames d'un caraté rouge provenant de l'Amérique centrale. La morphologie de ce dermatophyte le rapproche des *Bodinia*, des *Grubyella* et de l'*Endodermophyton roquettei* da Fonseca, 1925, agent du chimbéré.

Le rôle pathogène réel des champignons isolés des épidermomycoses polymorphes non spécifiques est assez difficile à préciser. Il n'y a aucun doute pour les dermatophytes proprement dits ou clostérosporés; ces champignons ne sont pas connus à l'état saprophytique et sont par conséquent toujours parasites et pathogènes, On peut admettre que les champignons qui se développent dans l'épiderme lui-même peuvent, dans certains cas, être considéres comme pathogènes. Quant à ceux qui vivent à la surface des lésions, dans les parties macérées et les enduits crémeux, leur rôle pathogène reste assez douteux. Il est extrêmement difficile de savoir si ce sont de simples saprophytes, trouvant un terrain favorable dans les exsudats qui se forment à l'abri des plis cutanés, en milieu chaud et humide, riche en matériaux nutritifs. Ceci est surtout vrai pour les blastosporés, dont la présence doit être souvent la conséquence et non la cause de la lésion.

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Section of Epidemiology and State Medicine.

President—Dr. S. Monckton Copeman, F.R.S.

[*April* 27, 1928.]

Post-Encephalitis and Its Problems.

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Abstract.—Attention to-day is chiefly focussed upon the consequences of encephalitis lethargica; the disabled victims of the disease present many problems, both medical and administrative.

The disease has a high case-mortality; though the majority of deaths occur during the primary attack, in many cases death occurs at much longer intervals after onset; the death-rate is estimated at 35 to 40 per cent. From the after-histories of some 3,000 patients in London, Glasgow, Belfast, Manchester, Bristol, Sheffield and other large towns, it appears that about 40 per cent. of notified patients become disabled. Generally speaking, severe sequels are preceded by severe symptoms in the original attack, but the Parkinsonian syndrome is a noteworthy exception to this rule.

Broadly classifying the sufferers from post-encephalitis, rather than the sequelæ observed in this condition, the author discusses (*a*) those who suffer mainly from physical sequels; (*b*) those who chiefly show origin of mental deterioration; (*c*) those who exhibit demoralizations.

The provision of suitable institutional accommodation for those with progressive physical disabilities (often combined with mental failing), the general re-training and education of the youngest victims and the training and control of adolescents with serious character-changes, constitute some of the problems of post-encephalitis.

While advocating further research the author considers that for the present the welfare of the passive helpless adult is best secured by *ad hoc* arrangements under the Poor Law Authorities or Board of Guardians. The youngest patients, with a more hopeful prognosis, need special institutional care and training on some such lines as those of the Encephalitis Unit at Winchmore Hill. It is hoped that the Mental Deficiency Act, 1927, will prove of great help for the management of moral delinquents.

(1) Introduction.

Ten years ago the condition now known as encephalitis lethargica, or epidemic encephalitis, was first recognized in this country. It has been defined as "an inflammatory disease of the brain, sometimes acute, at other times subacute or chronic, prolific in its clinical manifestations, disabling, if not fatal, in its results, difficult to diagnose and almost defiant as to treatment and prognosis."

Since then we have learnt something of the epidemiology of the disease: its sporadic occurrence, its comparative prevalence in the colder months of the year, its special incidence on adolescents, its impartiality for the sexes, its indiscriminate incidence in all social grades and its seeming independence of insanitary conditions. Much is still to be learnt about the way in which the disease is spread, its epidemiological relations with other diseases, and its relatively high incidence as reported in these islands compared with that abroad. The clinical zeal first brought to bear upon the strange syndrome is still needful since differential diagnosis may be difficult and the primary attack is often so slight that it escapes recognition.

Bacteriologists have taught us much concerning the histo-pathology of the disease and the character of the cerebro-spinal fluid, and something about the virus; there is much still to be revealed, however, about the ætiology of encephalitis lethargica. In the meantime attention is chiefly focussed upon its consequences and upon the question of how best to deal with those disabled by the disease.

One has to be careful about nomenclature, and I should explain that by "post-encephalitis lethargica" I mean that chronic phase of the disease which frequently succeeds the "acute" attack, and is marked by various symptoms, known as relics, residua, after-effects or sequelæ. I use the term "post-encephalitis" without prejudice to the possibility that the phase in question may be really part of a continuous infection of which the acute attack is the beginning.

The problems of post-encephalitis must be viewed in their right perspective. In 1926, encephalitis lethargica attacked only 0·06 per thousand of the population (the incidence-rate for scarlet fever during the same period was 2·1 per thousand). The case-mortality is certainly high but the toll taken of the general population in 1926 was only 34 per million living as compared with 961 and 1362 respectively for tuberculosis and cancer.

The disease accounts for less ill-health and disablement among the population than is attributable, for example, to measles, influenza, or venereal disease; but in proportion to the few attacked, there is probably no disease, with the possible exception of cerebro-spinal fever, whose death-rate and disabling-rate are both so high. Moreover, we do not as yet know of any preventable environmental condition or personal factor which disposes to the disease, nor is there any treatment known which will either influence the disease or ward off its sequels.

(2) The Serious Effects of Encephalitis Lethargica.

The following tables embody data supplied by the health authorities of some of the larger cities and towns in the United Kingdom.

(A) *Mortality.*—Table I shows the number of cases notified and deaths registered annually since 1919 together with the ratios of deaths to cases. The figures in

Table I.

Encephalitis Lethargica. Notified Cases and Registered Deaths, England and Wales, 1919-1927 (inclusive).

Year	1919	1920	1921	1922	1923	1924	1925	1926	1927	Totals
Cases notified	541	890	1,470	454	1,025	5,039	2,635	2,267	1,604	15,925
Deaths registered	294	480	729	339	531	1,407	1,372	1,325	1,155	7,632
Ratio of registered deaths to notified cases ...	54·3	53·9	49·6	74·7	51·8	27·9	52·1	58·5	72·0	47·9

the first two lines of the table are official in origin, but those in the bottom line will doubtless confirm the scepticism of many who distrust official figures. The notification figures do not include every case; indeed, it is probable that for every 100 cases notified there are another fifty which at first pass unrecognized. The mortality figures reflect errors in diagnosis and certainly include some deaths not caused by encephalitis lethargica. For both these reasons the ratio figures may be considered too high. On the other hand, the Registrar-General's net collects a good many bona fide deaths from the disease that escape the notice of those who saw the patients only during their primary illness.

An analysis of 452 death certificates for 1926, which give the interval between onset and death, showed that 28·8 of these deaths occurred from four months to six years after the onset of symptoms. A recent review, in 1927, of 259 cases notified in six Yorkshire towns, mainly since 1919, proved that nearly 60 per cent. of these patients had died; the results for each town were: Bradford (1921-1926), 97 cases, 59·8 per cent. fatal; Dewsbury (1924-1927), 13 cases, 61·6 per cent. fatal; Doncaster (1919-1927), 12 cases, 100 per cent. fatal; Hull (1919-1927), 100 cases, 61·7 per cent. fatal; Rotherham (1919-1927), 32 cases, 25 per cent. fatal; Wakefield (1919-1927), 5 cases, 40 per cent. fatal.

The fatality of encephalitis lethargica—if exaggerated by the rough ratio figures in Table I—is under-estimated in hospital and private records; I should say that it eventually kills, or helps to kill, 35-40 per cent. of those attacked.

(B) *Disablement.*—A certain proportion of those who die from the disease suffer previously from chronic or post- encephalitis, and a considerable number do not completely recover from the primary attack but suffer from after-effects. An attempt to estimate the percentage of patients who do not completely recover, and the disability following encephalitis lethargica, is shown in the next two tables.

Table II is concerned with the after-histories of 3,112 patients in large cities.

TABLE II.

FATE OF THOSE PERSONS WHO HAVE BEEN ATTACKED IN THE LARGE CITIES OF THE UNITED KINGDOM AND WHOSE CASE-HISTORIES HAVE BEEN SPECIALLY INVESTIGATED.

1	2	3	4	5	6	7	8	9	10	11	12	13
District or Town.	Period under review.	Date of investigation.	Total No. of cases traced and investigated.	No. of patients reported recovered.	Percentage of patients who recovered (of 4).	No. of patients with sequels not interfering with school or occupation.	No. of patients with sequels interfering with usual occupation.	No. of patients with sequels preventing occupation.	Total No. of patients with sequels.	Percentage of all patients with sequels (of 4).	Total No. of deaths.	Percentage deaths (of 4).
County of London	1919-25	1926	1,173 notified	373	31·8	61	100	180	341	29·1	450	39·1
			356 non-notified	39	11·0	14	45	158	217	60·9	100	28·1
Glasgow	1924	Dec., 1925	311	157	50·5	—	17	99	116	37·3	38	12·2
Sheffield	1924	1926	295	53	18·0	94	—	100	194	65·7	48	16·3
Manchester	1919-24	May, 1926	253	38	15·0	—	64	42	106	41·9	109	43·1
Lancs. County	1924	1925	227	44	19·4	29	26	49	104	45·8	79	34·8
Newcastle	1919-25	June, 1926	193	64	33·2		60		60	31·1	69	35·7
Bristol	1919-23	June, 1924	142	26	18·3	10	22	12	44	31·0	72	50·7
Belfast	April-June, 1924	Jan., 1927	162	13	8·0	8	29	84	121	74·7	28	17·3
			3,112	807	25·9	216	363	724	1,303	41·9	1,002	32·2

The sequels are grouped under three headings and in nearly every district the largest number of patients is found in the third group of serious sequels. The percentage of all patients suffering from sequels varies from 29·1 (London) to 74·7 (Belfast), while the mean sequel-rate for the eight districts is 41·9.

In Table III all the 2,711 cases reviewed in 1926 are grouped according to the years in which they were notified.

TABLE III.

SHOWS THE FATE (ASCERTAINED IN 1926) OF 2,711 PATIENTS, ARRANGED ACCORDING TO THE YEARS IN WHICH THEIR CASES WERE NOTIFIED.

1	2	3	4	5	6	7	8
Year in which illness began	Total number of cases reviewed	No. of patients now alive and apparently well	Per-centage	No. of patients now suffering from sequelæ	Per-centage	No. of deaths	Per-centage
1919	103	22	21·4	18	17·5	63	61·1
1920	217	36	16·6	75	34·6	106	48·8
1921	342	101	29·5	93	27·2	148	43·3
1922	91	24	26·4	18	19·8	49	53·8
1923	291	77	26·5	87	29·9	127	43·6
1924	1,291	436	33·8	507	39·3	348	26·9
1925	376	90	23·9	114	30·3	172	45·8
Total	2,,711	786	29·0	912	33·6	1,013	37·4

Here the sequel-rates generally are lower and the mean sequel-rate for the seven years is 33·6 per cent. The death-rates, however, are higher. Observe the ultimate fate of the 103 patients attacked in 1919.

Statistics for the peak year, 1924, show that for some of these districts the sequel-rate is unusually high and the mean rate 47·1; the mean fatality-rate for all the districts is low, viz., 23·8.

Not every patient who shows signs of post-encephalitis is prevented from following some occupation (though it may not be that followed before the attack), but the percentage of patients incapacitated from work or school is high, as will be seen from the Table below.

TABLE IV.

INCAPACITY RATES AMONG VARIOUS SERIES OF UNRECOVERED PATIENTS.

Series	Percentage of living unrecovered patients unable to work or attend school
All cases under review	56·2
London County cases	60·5
Lancashire County	47·1
Birmingham	80·5
Sheffield	51·5
Bristol	27·3
Manchester	39·6
Glasgow	85·3
Belfast	69·4

(3) FEATURES OF POST-ENCEPHALITIS.

The nature and comparative frequency of the sequels of encephalitis lethargica, as recorded by various observers, are shown in the following lists.

Dr. Phillips' list (Table V) refers to 93 patients of all ages treated for post-encephalitis at Southmead Hospital, Bristol, during 1924-5-6.

TABLE V.

SOUTHMEAD HOSPITAL, BRISTOL: COMPARATIVE FREQUENCY OF VARIOUS SEQUELS.

Nature of sequel	Percentage of unrecovered patients
Apathy, dullness, drowsiness and defective memory	28·0
Dementia	—
Viciousness	3·0
Irritability, loss of will power and other alterations of mentality	40·0
Inversion of diurnal rhythm	10·0
Squint	Ocular palsies
Ptosis	24·0 per cent.
Nystagmus	—
Pupils: defective reaction on convergence, irregularity	70·0
Diplopia	—
Paresis, facial	15·0
„ eyelids	35·0
„ arm and hand	15·0
„ leg	20·0
Tremor, tongue and lips	12·0
„ arm and hand	40·0
„ leg	5·0
Speech, stammering and slurring	10·0
Parkinsonism	40·0
Choreiform movements	—
Involuntary movements, abdomen, hands, arms	7·0
Constipation	—
Thirst	2·0
Hyperidrosis	8·0
Sialorrhœa	40·0
Spasmodic cough	—
Dysphagia	5·0
Severe headache	—
Jacksonian epilepsy	—
Incontinence of urine or fæces	5·0
Abnormal obesity	—

Table VI refers to 128 Glasgow patients whose histories after leaving hospital were also investigated by a single observer, Dr. C. M. Smith.

TABLE VI.

THE FOLLOWING IS A LIST OF SIGNS AND SYMPTOMS OBSERVED AND REPORTED BY DR. C. M. SMITH IN 128 PATIENTS, A YEAR AFTER THEIR PRIMARY ILLNESS IN THE BELVIDERE HOSPITAL, GLASGOW.

Sign or symptom	No. of cases	Sign or symptom	No. of cases
Insomnia	55	Tics	13
Asthenia	46	Pallor	13
Day drowsiness	31	Restlessness	11
Psychic changes	24	Headache	11
Nocturnal restlessness	21	Pareses	7
Parkinsonism	17	Neurasthenia	6
Pain	17	Constipation	5
Hyper reflexia	16	Adiposity	5
Hyperkineses	14	Speech changes (excluding bradyphasia)	6
Eye symptoms:—		Abnormal gait (excluding bradykinesia)	5
Paresis of accommodation	14	Nocturnal enuresis	3
Anisocoria	10	Absent-mindedness	3
Ptosis	5	Bulimia	2
Blurred vision	4	Furunculosis	2
Diplopia	3		
Squint	2		
Myosis	2		

Table VII concerns 134 selected cases of post-encephalitis (children) admitted to Winchmore Hill encephalitis unit between November, 1925, and December, 1926.

TABLE VII.

THE CONDITIONS OBSERVED AND THE NUMBER OF PATIENTS IN WHOM OBSERVED, AMONG CHILDREN WHO HAVE BEEN ADMITTED TO THE WINCHMORE HILL ENCEPHALITIS UNIT.

Character changes	86	Lack of expression	8
Sleep disturbance	78	Facial weakness	8
Mental changes	75	Speech defects	7
Parkinsonism	31	Muscular weakness	6
Emotional symptoms	15	Choreic movements	5
Restless and fidgety	13	Palsying limbs	3
Dribbling	12	Squint	6
Tremors and twitchings	11	Ptosis	2
Irregularity in gait	11	Irregular movements of eyes	1

On the whole, the signs and symptoms of post-encephalitis recall those of the primary stage of the disease; and, with a few important exceptions, serious sequels are usually anticipated by severe primary symptoms. This is the experience of Professor Hall. The relationship of sequels to previous symptoms is shown in Table VIII, which is adapted from the report of the Sheffield epidemic of 1924, published by the Medical Research Council. The exceptions are here illustrated.

TABLE VIII.

SHEFFIELD: SEQUELS IN RELATION TO PREVIOUS SYMPTOMS.

Disorder	During attack in 1924: No. of patients affected thereby	During attack in 1924: Percentage (of 301 traced cases)	After the attack (end of 1925): No. of patients affected thereby	After the attack (end of 1925): Percentage (of 254 traced survivors)
Ocular defects	215	71·0	72	28·0
Speech defects	—	—	Rarely noticed apart from Parkinsonism	
Vertigo	42	14·0	35	13·8
Pain (other than headache)	74	24·5	34	13·4
Moral effects	—	—	20	7·8
Mental changes	28	9·3	53	21·1
Disorders of sleep:—				
Insomnia only	66	21·9	Insomnia with or without lethargy, 24	80·0 (of those in which sleep disorder persisted)
Lethargy only	101	32·2	ditto 31	68·6 ditto
Lethargy and insomnia combined	85	28·2	ditto 28	82·4 ditto
Disorders of respiration	18	5·9	20	6·6
Parkinsonism	6	2·0	64	21·3

The table shows that moral effects, mental changes and Parkinsonism mainly belong to the aftermath of encephalitis lethargica. With Parkinsonism it is often extremely difficult to get any definite history of an introductory illness.

(4) Types of Post-Encephalitis Patients.

The great variety of sequels invites classification, but from the administrative point of view it is more useful to classify the sufferers than the sequels; I therefore group the former into (*A*) those with mainly physical sequels, (*B*) those who chiefly show mental deterioration, and (*C*) those who exhibit demoralizations. This grouping is loose; a Parkinsonian patient, for instance, may partly qualify for two or more of these groups, or a patient may pass from one group to another at different stages of his post-encephalitis career.

Group A.—Among physical sequels none is so well known as Parkinsonism; indeed this syndrome is so common and so important that it is regarded by some as almost synonymous with post-encephalitis. It is difficult to correlate the statistics of different reporters whose criteria in defining Parkinsonism vary considerably, but it seems that about 25 per cent. of those attacked, roughly half those who manifest after-effects, sooner or later—generally later, develop Parkinsonism. Most cases originate within three to eighteen months of the primary attack, but in a few instances the latent period has been prolonged for two, three, and even five years. In contrast with paralysis agitans, post-encephalitis Parkinsonism affects both sexes about equally, and by no means selects the old. For instance, the records of 557 unrecovered patients received from London, Manchester, County of Lancashire and Bristol show that 22·7 per cent. of the Parkinsonians were under 16; other records show that a substantial majority of Parkinsonian patients are under 30. The appearance of Parkinsonism is regarded by most as ominous; in most cases the patient gradually becomes helpless and unfitted for work; physical, and often mental, deteriorations occur in the hopeless cases, which may end in severe asthenia. While some patients appear placid and resigned, others are acutely depressed. Dr. Phillips, of the Southmead Hospital, Bristol, considers that Parkinsonian patients are particularly prone to suicidal impulses. A few instances are recorded of patients recovering from severe Parkinsonism sufficiently to resume their occupation, but the condition usually ends fatally, and accounts for most of the late deaths from encephalitis lethargica.

There are many sequels of clinical interest—some are included in the Parkinsonian syndrome—which might be considered with the physical manifestations of post-encephalitis, but I merely mention such conditions as residual palsies, tremors, abdominal myoclonus, obesity, enlargement of the thyroid gland, and strange respiratory disturbances.

Group B.—A noteworthy characteristic of encephalitis lethargica is that it often either leaves its victims with mental sequels of the original attack, or prepares the ground for subsequent mental disorders, and it is probable that the mental processes of every subject of the disease are affected, either in the primary attack or subsequently. From Table IX the proportion of sequelæ among which mental symptoms appear works out at 27 per cent; this is probably below the mark, since in most records Parkinsonism—which is frequently associated with mental disturbances—is considered separately. In the Sheffield outbreak, mental symptoms remained

Table IX.

Incidence of Mental Sequels among Unrecovered Patients.

Series	No. of unrecovered patients	No. showing definite mental impairment	Percentage
Ministry of Health, 1919-20	250	57	22·8
Lancashire County, 1924	104	27	26·0
Bristol, 1921-23	44	18	40·9
Newcastle, 1919-25	60	17	28·3
Birmingham, 1924	183	41	22·4
Sheffield, 1924	135	53	39·3

in 53·6 per cent., and in the Lancashire series in 23·4 per cent. of patients originally affected mentally; further, in the same two series of cases, mental symptoms first appeared as sequels in 14·9 per cent. and 16·0 per cent., respectively, of the survivors.

Post-encephalitic patients with mental symptoms may broadly be divided into the excitable and restless, and the depressed. Typical of the irritable or positive type of mental disturbance is nocturnal restlessness associated with the inverted sleep rhythm so common in children. In lists of post-encephalitis sequels in children this disorder always appears first or second in order of frequency and probably occurs in 76 of every hundred cases of juvenile post-encephalitis. Fortunately, with time and appropriate treatment the condition generally clears up.

A different picture altogether is presented by the passive or depressed type of patient. Inability to make any progress at school is due to dulled intelligence, impaired memory and perhaps drowsiness, rather than to the restlessness, impatience and lack of concentration which make teachers despair of the excitable type of child. Children in an advanced stage of Parkinsonism provide examples of the passive type of case, but it is mainly adult patients who suffer from mental anergia. The mental disturbance in both types of case may be severe enough to justify certification; the irritable, mainly for the sake of others, are admitted to institutions as suffering from hallucinations, with homicidal tendencies, or mania; while the depressed, mainly for their own sake, are admitted by reason of melancholia, suicidal impulses or complete dementia. Up to the end of 1927, 674 encephalitis lethargica patients have been certified and admitted to mental hospitals and institutions either during the acute attack or in the post-encephalitis stage: this represents 5 per cent. of all notified patients, including those admitted during their primary illnesses.

Group C.—I borrow from Dr. Mapother the word "demoralizations" to characterize the changes in patients of this group. I have, however, taken the word from its original context, where it is included among mental defects. To speak of demoralizations or conduct changes is almost to speak of post-encephalitis in children; but the intellect in such children is often unimpaired, and in fact may often appear strangely bright. Many of these highly emotional and troublesome children might well come within the following description by Dr. Tredgold:—

"The moral imbecile is not lacking in the capacity for acquiring school knowledge as are all ordinary imbeciles and a large number of the feeble-minded. On the contrary, he is often possessed of a cleverness, even a brilliance, which distinguishes him very widely from most ordinary defectives. He is also usually a good conversationalist, is ready at repartee and nimble-witted; he has an engaging manner, and is an exceedingly plausible and ready liar. But he is absolutely devoid of all moral and altruistic feeling. He will lie when the truth would answer his purpose just as well. He knows neither shame nor gratitude, and will requite the utmost kindness and consideration with heartless robbery; in doing which he will not even refrain from violence. At the same time he is so devoid of the capacity for mental comparison and discrimination, for forming judgments and for looking ahead—in short, of those attributes of mind which collectively constitute wisdom or common sense—that he is quite unable to appreciate the personal disadvantages of such conduct. Even in the commission of his crimes he will either neglect the most simple and obvious precautions, or he will act with such a total disregard of prudence as to make detection inevitable."

Such a character is known among juvenile post-encephalitics, and in departmental files and medical journals are graphic narratives of these wayward children and their vagaries. Dr. Shrubsall considers that these character changes originate "in the state of irritability, lack of inhibition, and consequent impulsiveness which immediately follow the acute phase of the illness and become fixed, either by

habit formation or by secondary development of psycho-neuroses, dependent on the particular features of the individual environment." Whatever the cause, the disposition is always changed for the worse.

(5) THE PROBLEMS OF POST-ENCEPHALITIS.

It will be convenient, I think, to discuss the difficulties of post-encephalitis in relation to two classes of individual—(*a*) those whose illness occurred before the age of 18 and (*b*) those whose illness took place after that age. Consider first a young school-child, who returns home after several weeks or months in hospital. Released from the discipline of institutional life, with a disposition strangely altered, and suffering from nocturnal restlessness, the child soon becomes unmanageable and a terror to his family. Or it may be that the child is already in the grip of Parkinsonism, and is teased by brothers and sisters, or scolded by parents for a slowness of mind and body they do not understand. In both cases life at home is not suitable for these children. It may be, however, that someone else's home may prove not only suitable, but beneficial, and a certain number of children have undoubtedly benefited by being boarded out: but there is a difficulty in finding foster parents willing and fitted to take such children. The Guardianship Society at Brighton concerns itself with finding suitable homes for mentally or morally affected children, and has certainly proved helpful in the case of some post-encephalitis children.

It is often difficult to decide whether the advantages of keeping a post-encephalitis child working as far as possible among normal children are not cancelled by the disadvantages to the other children, due to the presence in their midst of one or more pupils whose conduct and disposition have become abnormal. Generally speaking, except in the case of those children with only faint traces of their previous illness, a return to their usual school is not practicable. For some, the schools for backward and mentally defective children will provide the extra individual attention and patience necessary to re-establish the diminished power of concentration and arrested intellectual development. Unfortunately, it is impossible to admit children showing conduct changes to schools where their behaviour imposes an unjustifiable strain on the teachers, and may result in physical or moral harm to other children.

The resources of the education authorities will then be exhausted. In these circumstances an Encephalitis Lethargica Unit, like that at Winchmore Hill, supplies a want. Special arrangements are made for carrying on the child's education while under medical treatment and institutional discipline. Otherwise the patient grows beyond school age, and becomes more wayward than ever, and quite unmanageable at home. Then one day he steals a bicycle, is brought before a magistrate's court and put upon probation; this is soon broken, and he next appears in the court on a charge perhaps of assaulting a small girl. If between 12 and 16 years, he may then be committed to an industrial school or to a reformatory. From February 1, 1920, to January 31, 1927, 108 children of the type we are considering were committed to these Home Office Schools. Of the fifty-seven discharged, fifty-four (95 per cent.) showed no permanent improvement; of forty-nine post-encephalitic children remaining on January 31, 1927 (two died), ten were considered unsuitable for further retention. Indeed, "all the evidence serves to show convincingly," says Dr. Culverwell, "that attempts to train such children in Home Office Schools are not justified by the results obtained." In any case, committal to Home Office Schools is for a limited period only, and the young encephalitic—if he has not in the meantime come under the Board of Control—must eventually be released and either placed in employment, sent home, or sent, under the Released Persons (Poor Law Relief) Act, 1907, to the care of the Guardians. Up to the end of 1927, the post-encephalitis delinquents usually had no difficulty in avoiding the Board of Control, because many

of them were not considered mentally deficient, and because where there was undoubted deficiency it had not existed from birth or early age, and the patient could not therefore be certified according to the literal interpretation of the Act.

Though action under the Mental Deficiency Act, 1913, was carefully considered for every one of the 108 children admitted to Home Office Schools, less than a fifth of them could be so dealt with. With the passing of the amended Mental Deficiency Act last year, however, the technical difficulty of the principal Act was removed, and this, with the new definition of "moral defectives" should prove helpful in dealing with troublesome post-encephalitics. In the majority of cases the disposal of the patient in a suitable Mental Deficiency institution is probably the best course. Obviously the youth, now older and more incorrigible, is less than ever suitable for his home environment; and if placed in employment he seldom remains for long where he is put. For a time he may be tolerated in a Poor Law institution if he consents to remain there; but the Guardians have no power to retain him against his will unless they adopt him, and this can only be done if he is under 18. It is during the periods variously spent at home, in and out of occupations, and after discharge from Poor Law institutions that the ex-reformatory school inmate usually comes into more serious contact with the law and the prison authorities. A few offenders have been sent to Borstal Institutions, but according to the Director of the Borstal Association, which keeps in touch with boys and girls discharged from such institutions, the post-encephalitis cases have shown little or no improvement. Lastly, our troublesome individual may be committed to prison, where between April 1, 1925, and March 31, 1927, seventy-two of his kind have found a temporary home. The Medical Commissioner has reported that of the seventy-two prisoners "fifty-four showed either no harmful effect from imprisonment or were definitely improved; the remainder deteriorated." Dr. Griffiths concludes that about two-thirds of these seventy-two cases might have been dealt with under the Mental Deficiency Act of 1927.

Now consider the adult who has returned home after a prolonged illness in hospital. Not so quickly perhaps as with the school-boy, the family realizes that the returned patient has changed in various ways and does not easily resume his accustomed rôle. There may be aimless wandering about the house in which the sufferer does not feel at home and has apparently lost interest. This mental anergia may develop until the sufferer becomes a confirmed melancholic. In other cases there may be anxious and laborious efforts to resume the occupation upon which the welfare of the family normally depends. The father, perhaps an excellent workman before his illness, is taken back by his employer and tries to carry on as before; but lost initiative, impaired memory, slowness and reduced physical powers prevent him from justifying the experiment. He tries other jobs with no better success and in the intervals of unemployment he broods at home and bemoans his fate. Or it may be the wife, once the intelligent mainstay of the home, who has now become careless and lethargic, unmindful of her children, and generally incapable of attending to her household duties. These older patients do not, as a rule, come in contact with the law nor do they excite the same public interest as juvenile cases. Nevertheless, they present very difficult problems within their own domestic circle, and when the powers and patience of their relatives reach breaking point their removal from home becomes urgent. But there is often much difficulty in finding a suitable refuge for such patients. They may remain uncertifiable for a considerable time during which they may be accommodated for brief periods by relatives or through other temporary arrangements; either they themselves are incapable of applying for admission to a mental institution as voluntary boarders, or their relatives will not consent to such a step; similarly, accommodation in the Poor Law Infirmary is not welcomed, while such cases are too chronic and hopeless for hospitals and convalescent

homes. Eventually a certain number, through melancholia, suicidal impulses or general

TABLE X.

ADMISSIONS OF ENCEPHALITIS LETHARGICA PATIENTS TO MENTAL INSTITUTIONS, 1918-1927.

Admissions in 1918-1925 / 1926 / 1927 / Totals	Mental hospitals	M.D. institutions	Mental hospitals	M.D. institutions	Mental hospitals	M.D. institutions	Mental hospitals	M.D. institutions	
Up to age 17	39	54	36	47	47	32	122	133	255
17 to 21	36	13	32	19	61	14	129	46	175
Over 21	39	11	54	4	103	3	196	18	214
All ages	114	78	122	70	211	49	447	197	644

insanity, often associated with Parkinsonism, become certifiable and find asylum either in mental hospitals or in the mental wards of Poor Law institutions specially approved by the Board of Control. Table X gives details concerning post-encephalitis lethargica patients admitted to mental institutions up to the end of 1927.

In considering how the difficulties connected with after-care and control should be, and to some extent are, being met, I subdivide the juvenile group into those below and those above the age of puberty. The youngest sufferers need special care and homely discipline, preferably at the hands of women, by whom their elementary education will also be carried out. For such, an institution similar to that at Winchmore Hill provides some hope of improvement for less demoralized children and considerable alleviation for the Parkinsonian patient. Those who have reached puberty often prove difficult or dangerous as institutional companions for the youngest patients; their exuberant emotions and greater potentialities for mischief need such outlets as are provided by vigorous outdoor occupations and by technical instruction in suitable forms of craftsmanship; they are best under male supervision. At present no special provision has been made for this class, though it has been considered in connexion with the Unit at Winchmore Hill. Some of these difficult adolescents, however, will in future be dealt with under the Mental Deficiency Act of 1927. The adult patients, as a class, do not require so much re-education and control as relief from anxiety and expert attention to their physical needs. For a large majority of such the Poor Law institutions provide a refuge. In Bristol, Glasgow and a few other places, the local Guardians have recognized the needs of post-encephalitis patients and, in collaboration with other local bodies, have made special provision for them. Dr. Phillips' post-encephalitis section at the Southmead Hospital, Bristol, is of special interest, because it ministers to the medical, educational and recreational needs of both juvenile and adult patients and accepts all types of patient except the certifiable. The Board of Control has also realized that patients whose mental symptoms are due to encephalitis lethargica need particular study; provision for such patients is made at the West Park Hospital, Epsom, and at four or five other mental hospitals in the provinces.

Discussion.—Dr. S. MONCKTON COPEMAN (President) said that Dr. Parsons' paper, though exceedingly interesting, was somewhat depressing in view of what he had to say as to the fatality of the disease, and as to the terrible mental and physical disabilities which so frequently follow in its train. He had, however, rightly stressed the point that the problems of post-encephalitis must be viewed in their right perspective; the toll taken of the general population in 1926, for instance, was only about one-thirtieth of that from tuberculosis, while the incidence-rate was less than one-third of that from scarlet fever in the same year.

He (the President) was specially interested to hear of the public-spirited action of the Bristol Guardians in establishing the post-encephalitis clinic administered by Dr. Phillips at their Southmead Infirmary, because it was at this Infirmary that he (the speaker) had some years ago been afforded the opportunity of carrying out the first official investigation in this country of the value of the Schick test and of toxin-antitoxin immunization respectively.

With regard to the success apparently obtained in the treatment of general paralysis of

the insane, more particularly of the mania type, by infection with a benign type of malaria, further trial of treatment on similar lines might be found of service in post-encephalitis. This seemed not improbable as no specific action was involved in its use in the treatment of general paralysis of the insane. In all probability a non-specific raising of resistance to the infection, on the part of the cerebral tissues especially, was responsible for the results obtained.

Dr. F. C. SHRUBSALL said that some figures collected during the last three years in London were of interest. The fatality-rate in confirmed cases over the years 1919—25 had been 37 per cent. The maximum incidence had been between ten and twenty with a slight preponderance of males over females, more especially at the younger ages. The greatest fatality-rate was at ages under 5 and the least at 10 to 15. The results of the work of the Encephalitis Unit at Winchmore Hill had been set out by the Superintendent, Dr. Borthwick, in his reports to the Metropolitan Asylums Board, which indicated much relief for individuals, but that very many seemed likely to need permanent care. An analysis of 403 cases not treated at the Unit showed that 220 had been notified and 183 not notified, so that nearly as many passed unrecognized at the initial stages as were recorded. Of this 403, 176 appeared to have recovered, though there was always the risk of a relapse, and 227 were still affected; of these 76 were paralysed or Parkinsonian and 105 mentally or morally affected.

The new Mental Deficiency Act, 1927, had minimized the difficulty with regard to early age; the new problem was whether moral affection alone would allow a subject to be deemed feeble-minded. If an attempt was made to certify a subject as morally defective it was necessary to show the care required was for the protection of others, care under this definition for the sake of the subject himself was specifically excluded by the Act. Before a child between 7 and 16 could be dealt with as feeble-minded, unless he was charged with an offence punishable in the case of an adult by imprisonment or rendering him liable to be sent to an industrial school, the consent of the Board of Education was necessary. Administrative officers were waiting with interest to note whether the Board would allow the notification of children with conduct disturbance and incomplete control who nevertheless had average intellects and attainments. If not, the value of the new Act would be diminished at the age at which the children could most benefit from careful treatment. A child too greatly disturbed in conduct to remain at a unit needed care in a mental hospital or certified institution. He trusted no difficulties would arise and that cases could be passed through rapidly to permit of continuity of care.

Dr. J. D. ROLLESTON said that although his clinical experience of lethargic encephalitis had not been extensive, he had kept in touch from the first with the literature which he had compiled on several occasions[1]. When the disease first appeared in this country it had been mistaken for botulism, but at a special discussion on this subject by the Section for the Study of Disease in Children, he (Dr. Rolleston) had suggested that the cases so described were really examples of encephalitis.[2] In collaboration with Dr. H. Morley Fletcher[3] he had been one of the first in this country to draw attention to the characteristic association of diurnal somnolence and nocturnal restlessness to which Dr. Parsons had alluded.

Referring to ætiology, several writers,[4] Lépine in particular, had emphasized the importance of a nervous predisposition, but this view did not appear to have received general acceptance, especially as the disease was not very uncommon among doctors. With regard to treatment, lethargic encephalitis appeared to be one of the "opprobria medicinae." A multitude of remedies had been suggested but their efficacy was doubtful, and some, particularly fixation abscess, were dangerous. Specific treatment in the form of a vaccine had been employed by some French observers[5], some years ago, but he (the speaker) had not found any reference to it in recent literature.

Dr. GRAHAM FORBES asked what was the evidence of relationship between the type of the initial attack, i.e., its mildness or severity, and the development of post-encephalitis and its degree of severity, and whether, in a quite mild, or even doubtful case there was sufficient experience to justify a forecast of the probability of post-encephalitic

1 *Medical Science: Abstracts and Reviews*, 1919-20, i, 87-92; 1921, v, 193-210; 1923, vii, 443-451. *Medical Annual*, 1925, 129-32; 1926, 140-2; 1927, 136-7; 1928, 138-41.

2 *Proceedings* (Sect. of Dis. in Child.), 1918, xi, 52.

3 *Brit. Journ. Child. Dis.*, 1921, xviii, 69.

4 *Medical Science: Abstracts and Reviews*, 1921, v, 193-4.

5 *Medical Annual*, 1925, 132.

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sequelæ. Were there any indications of a familial susceptibility to the disease? He had known of its occurrence in a brother and sister, not in contact with each other. The sister's attack proved fatal during the absence of her brother in India. On his return to England the brother fell a victim to the disease, but in spite of the severity of the attack, with left hemiplegia, he made a complete recovery, and was now apparently in good health and engaged in medical practice.

Dr. N. GRAY HILL said that in the autumn of 1925 he had observed a case of post-encephalitis treated by malaria. The acute attack of encephalitis lethargica had occurred in 1921 and was very severe. The onset of Parkinsonism was insidious but steadily progressive. As an immediate result of the malaria the patient became distressed and helpless and the Parkinsonism more marked. When the malaria was terminated the general condition improved and it was then thought that the treatment had been beneficial; the patient soon, however, returned to the condition in which she had been before the malaria treatment, and had since become more and more incapacitated.

Dr. D. SAGE SUTHERLAND said that during 1924-25 about 100 cases certified as encephalitis lethargica were admitted to Monsall Hospital under his care. While the diagnosis in the majority of cases presented little difficulty, a small number exhibited no abnormality on admission, but gave a history of transitory vertigo and diplopia. Definite diagnosis was very difficult. In the subsequent history of such cases slight attacks were found in some instances to give rise much later to post-encephalitic symptoms, including Parkinsonism, and in his experience the incidence of post-encephalitic symptoms had no definite relation to the severity of the original attack. Cases of Parkinsonism had improved considerably in hospital by removal from home, careful dieting, open-air treatment and ultra-violet ray baths, though a cure had not been affected.

A case which presented some analogy to the treatment of general paralysis of the insane by inoculation with malaria was that of a girl aged 14, admitted last year with well-marked Parkinsonism, salivation, inequality of pupils, tremor, vertigo and headache; after three months in hospital she contracted a severe attack of typhoid fever, her temperature ranging from 103° to 104° F., and pyrexia lasting about twenty-eight days. On recovery from typhoid very marked improvement was noted in the post-encephalitic symptoms. Salivation had entirely disappeared, clonic spasms of the arm were absent, tremor was completely absent, and her general health and mentality were considerably re-established. She could walk more firmly, and was discharged three months later in a much improved state of health.

Dr. W. S. C. COPEMAN asked why only those suffering with the mental sequælæ of encephalitis lethargica should receive treatment from central authorities, in view of the fact that mental changes only constituted about 30 per cent. of the various late manifestations of the disease, and treatment was probably of more avail actually in the other 70 per cent.

Dr. G. CLARK TROTTER said that the paper was interesting to him personally because he had to investigate and report on a fair number of cases. The inquiries had to be made by sanitary inspectors, for whom he simplified matters by translating the technical terms into common language. The inspector endeavoured to see a relative apart from the patient, and get a general idea of the case. Unless they were interested by being given lucid information, the inspectors might think the work uninteresting, and do it indifferently.

Dr. Parsons' paper had brought out some very interesting information, which it would be well to remember in going over the sanitary inspectors' reports.

With regard to the case the patient whose post-encephalitic condition had benefited from an attack of typhoid fever: probably many Members would have noticed that an attack of typhoid fever had been apparently beneficial to a patient whose health had previously been indifferent; it seemed as if the attack of typhoid fever had some specific action, and that the benefit was not due to long rest and treatment.

Dr. PARSONS (in reply) said that it was precisely the distressing cases which gave rise to administrative problems and that such cases were not limited to persons with mental symptoms. He had no knowledge of the infection of the disease through food and could not find that the individuals in any particular calling were peculiarly susceptible; instances of familial infection were well known, but were not common. Treatment by induced malaria had not apparently proved very promising; the case of improvement following enteric fever recalled the cases of two Parkinsonian patients who were apparently cured of this syndrome by an inter-current attack of acute appendicitis.

Section for the Study of Disease in Children.

President—Mr. GEORGE E. WAUGH, F.R.C.S.

CASES SHOWN AT THE MEETING HELD AT THE LONDON HOSPITAL, APRIL 27, 1928.

Facial Nævus: Tumour of Pons, ? Angioma.

By W. RUSSELL BRAIN, M.D.

History.—E. G., aged 11 years, has had a nævus on the right side of the face since birth. She was in good health until November, 1924, when she began to suffer from headache and vomiting. These symptoms were severe for about six months and then disappeared until six months ago. Unsteadiness of gait has been conspicuous since 1924. Her father thinks that her gait has never been normal and that she has always staggered somewhat and dragged her right leg. She has had diplopia occasionally but there has been no subjective visual impairment. She gained weight rather rapidly at the end of 1925. No other illness except measles. Family history unimportant.

Present Condition.—She is a plump girl (weight 4st. 13lb.) with a nævus ("port-wine stain") over the right side of the forehead, right cheek and right side of upper lip. Vision: right eye $\frac{6}{24}$, left eye $\frac{6}{12}$. The visual fields are full. Fundi: slight secondary optic atrophy. The left pupil is larger than the right; both react well to light and on convergence. There is weakness of both external recti, the left being more affected than the right, but no diplopia. Nystagmus is present on lateral fixation to right and left. Bilateral weakness of the lower part of the face for voluntary movement, more marked on the right side. The soft palate moves poorly and the tongue is protruded to the left.

In the limbs, voluntary power is fair. The upper limbs show slight ataxia, especially the right, and marked adiadokokinesia. There is hypotonia of the upper and moderate spasticity of the lower limbs. The tendon reflexes are present and equal in the upper limbs, exaggerated and equal in the lower limbs. The right plantar is extensor and the left doubtful. There is no sensory loss. Gait somewhat ataxic.

Radiograms of the skull show slight "convolutional thinning."

There is a possibility of the intracranial tumour being an angioma. It is well known that angioma of the brain may be associated with angiomata elsewhere. In a small number of cases there have been angiomata of the face in the area of distribution of the trigeminal nerve; Cushing has described three such cases. In those, the cerebral angioma apparently arose in the dura and involved the cerebral cortex of the same side, and all the patients had epileptiform convulsions and hemiplegia. With regard to subtentorial angiomata, Lindau has described twenty-four cases in the cerebellum, four in the medulla. In 20 per cent. of cerebellar angiomata there was also angioma of the retina. I do not know of any cases of subtentorial angioma associated with angioma of the face.

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Mr. HUGH CAIRNS said this case raised a very important point for the surgeon, who when intending to operate for intracranial tumour on a patient who had a cutaneous angioma of the face, must be prepared for severe bleeding, for in most cases the intracranial tumour would prove to be also an angioma. In these cases one might even have a death on the table from violent hæmorrhage when the osteoplastic flap was reflected. That almost happened with Cushing's first case of this kind. In the usual type of intracranial angioma, preliminary treatment with X-rays was of value since it seemed to produce a thrombosis in the vessels of the tumour. He had seen a man with intracranial angioma who had had fits for forty years. There had been an attempt at operative removal, but there was so much bleeding when the bone flap was reflected that operation was abandoned. The tumour was then treated by X-rays and when a second operation was subsequently done it was found to be quite thrombosed and was easily removed.

Multiple Subcutaneous Abscesses.

By W. J. O'DONOVAN, M.D. (shown by Dr. R. T. BRAIN).

S. F., AGED 4 months.

On Examination.—Pale but bright child with small nodules on right knee, abdomen and buttock. Skin over first two nodules reddish, purple and shiny, but normal over nodule on buttock. Nodule aspirated.

Clinical Report.—"Degenerate pus cells and débris. No organisms seen. Culture, sterile."

Treatment.—Light therapy.

Dr. LEOPOLD MANDEL said he had had three cases of the kind, and in each of them *Staphylococcus albus* was grown. They were treated with very small doses of autogenous vaccine and light; in these cases the tungsten arc was used, though probably they would react equally to the mercury-vapour lamp.

Cirrhosis of the Liver.

By R. CECIL WALL, M.D. (shown by Dr. G. KOHNSTAM).

J. C., MALE, aged 6 years.

History.—In November, 1927, when he first came under observation, the child had an attack of vomiting which lasted three days: he also complained of peri-umbilical pain and the mother noticed that his abdomen was swollen.

Past History.—Pneumonia three and a half years ago.

Family History.—No history of tuberculosis obtained.

Habits.—Child said that he shared drinks of gin with his father on many occasions.

On Examination.—(November, 1927.) No pyrexia; œdema of abdominal wall; no œdema of feet; no jaundice; tongue clean. Cerebro-spinal fluid: clear. Respiratory system: dullness of both lung bases due to high diaphragm. Abdomen: marked bulging in flanks and umbilicus; shifting dullness and fluid thrill obtained; lower border of liver felt by dipping two fingers' breadth below costal margin. Spleen not felt. Nervous system: pupils regular and react to light and accommodation. Knee-jerks obtained.

After one week in hospital the ascites disappeared. Wassermann reaction was negative. Blood-count: leucocytes 10,400; hæmoglobin 80 per cent.; colour index 0·78.

The child is now in apparently good health though he is not gaining weight. The liver is still 1½ finger's breadth below subcostal margin, is firm in consistency and has a moderately sharp lower border.

Dr. F. PARKES WEBER said he thought the evidence of alcoholism should be again carefully investigated in this case. The cases of hepatic cirrhosis in children that he had seen had apparently not been due to alcoholism, but some cases had in the past been described that seemed to be really alcoholic in origin. The hard liver, extending a little below the costal margin, in the boy now shown, corresponded to what was often found in cases of alcoholic cirrhosis in adults, in a quiescent stage, when the liver cells were functioning well, though a considerable degree of hepatic fibrosis had already developed.

Carcinoma of the Suprarenal.

By G. E. NELIGAN, F.R.C.S. (shown by Mr. V. DIX).

F. M., FEMALE, aged 5 years.

History.—For about six months the mother had noticed an increase in size of the child's abdomen. This had been more conspicuous during the two months before admission. The child had complained occasionally of abdominal pain. She had appeared also to be growing up rather rapidly.

Previous History.—Negative.

Family History.—Negative.

On Admission.—(February 16, 1928.) The child looked several years older than her age. There was great distension of the abdomen and the left side was filled by a hard fixed smooth tumour. The tumour was dull on percussion and did not move with respiration. There was no enlargement of the liver. The pubic hair was well developed.

Progress.—On April 27 Mr. Neligan performed a laparotomy. A tumour was found to be fixed to the posterior abdominal wall and also to the upper pole of the left kidney. The tumour and the left kidney were excised. The child has made an uninterrupted recovery.

Pathological Report.—Solid polygonal and giant-celled carcinoma of suprarenal.

[Specimen shown.]

Discussion.—Mr. G. E. WAUGH (President) said that there was one point about this case which he raised with hesitation because he was not present at the operation. The child seemed to be in an extraordinarily good state of health, and that was strange when a carcinoma of the suprarenal had reached such a large size. Looking at the specimen without being able to handle it, it appeared to him that it did not seem to have the relationship that an adrenal tumour—especially when the tumour was of a malignant nature—should bear to the kidney. With real adrenal tumours of such a size one could not separate them from the upper pole of the kidney. In the present case the kidney was symmetrically arranged with regard to the whole of the growth, and fell into the category of hypernephromata. He did not know whether those who saw the operation would regard that as an alternative explanation.

Mr. HUGH CAIRNS said he saw this child before operation and she was, at that time, decidedly cachectic. Looking at the case casually it then seemed that there could only be one outcome of operation and that an unfavourable one. After the surprising success of the operation there had been an amazing change for the better in general health.

Achondroplasia in the Third Generation.

By DONALD HUNTER, M.D.

JENNY DORFMAN, III 5, female, aged 13 years.

The mother, II 6, is a short woman (52 in.) of normal proportions (fig. 1). She was born in Poland. In 1914 she was admitted to the London Hospital (Dr. Maxwell) as a full term primigravida with a small pelvis (interspinous, 8¼ in.,

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intercristal, 9½ in., diagonal conjugate, 3¾ in.). The child, III 5, was delivered after a difficult labour (twenty-eight hours) and weighed 4½ lb. She was breast-fed till 18 months, at which time she was treated for rickets, and wore a poroplastic jacket for some time. She had her first tooth at 6 months and walked just before the age of 3 years. At 7 she was sent to a special school of the L.C.C. She is very intelligent and reads and writes well.

Height 37½ in. (fig. 1). Head and face disproportionately large for height. Circumference of head 20½ in. Bridge of nose depressed and broadened, nostrils

FIG. 1. (Photograph taken in 1925.)

rather wide. Looks much older than her age. The limbs are short and the shortening affects the proximal more than the mesial segments. Distal extremities of fingers extend only to great trochanters. The fingers show an approximation to equality in length, but the trident hand is not present. Lower ends of radius and ulna prominent. Gait, "waddling." The soft tissues of the limbs seem to be increased so that the skin folds, especially on the thighs, are exaggerated. Upper end of tibia broad and prominent. No beading of ribs. Lordosis with prominent abdomen and buttocks. There is also a lower dorsal kyphosis which is not rigid. No further abnormality found in chest, abdomen, or nervous system.

Radiograms.—Shortening of base of skull with a large calvarium. Humerus and femur much shortened. Diaphyses widened at ends of long bones and epiphyses enlarged, especially at upper end of tibia and lower end of femur. Left humerus and left radius curved; other long bones straight. Dorsal spine normal. Small pelvis, wide sacro-iliac synchondrosis and deep acetabula. (Dr. G. E. Vilvandré.)

The father, Leon Dorfman, II 7, is a typical achondroplasic (fig. 2, right). He was born in Russia. He is 38 years of age and has earned his living on the stage since the age of 10. He is very intelligent, muscular and strong. Height 47 in. Depressed bridge of nose. Circumference of head 22 in. His hands show fingers

FIG. 2. (Photograph taken in 1911.)

approximately equal in length. In extension the distal extremities of the second and third fingers are widely separated (fig. 3).

The father had four sisters apparently of normal stature. II 1, died at the age of 21 years. II 2, died in childbirth at the age of 29 years, leaving four healthy children of normal stature (III 1, 2, 3, 4). II 4 and II 5 were of normal proportions: they died as young children.

The paternal grandfather, Ossif Dorfman, I 1, measured 44 in. in height (fig. 2, left). He was born in Russia. Photographs leave no doubt that he was an achondroplasic. He worked all his life on the stage and died in 1913 at the age of 72 years.

The paternal grandmother, I 2, according to descriptions, was rather short but of normal proportions.

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Comment.—Writing in 1912, Rischbieth and Barrington [1] stated of achondroplasia that: "It does not seem feasible to determine, at the present day, the proportion between 'hereditary' and other cases." They recorded eighty pedigrees,

FIG. 3. (Photograph in 1924.)

of which twenty showed cases occurring in two or more generations. Their series of cases suggests that the female sex is more predisposed than the male, for they found amongst 126 cases, 70 females and 56 males. Kassowitz [2] found amongst 29 cases, 25 girls and 4 boys.

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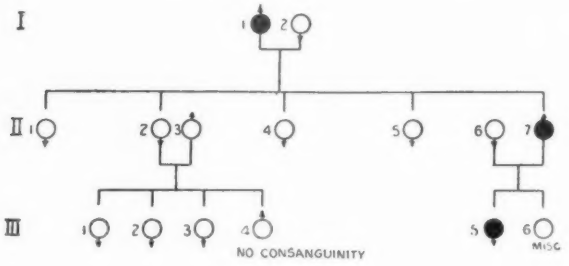

Case for Diagnosis.

By A. G. MAITLAND-JONES, O.B.E., M.C., M.D.

E. J., FEMALE, aged 11 months.

History.—Full-time child, normal labour. Nothing unusual was noticed until the infant was four months old. She then began to have fits which have continued, at infrequent intervals, up to the present time. The fits consist of jerking movements of the limbs with rolling of the eyes.

Family History.—Father and mother alive and well. One other child alive and well. No history of a similar condition amongst the parents' relatives.

Present Condition.—Apparently both blind and mentally defective. Slight spasticity of the lower limbs. Tendon reflexes brisk. Pupils react to light. Examination of the fundi shows primary optic atrophy. Cerebro-spinal fluid is normal, the Wassermann reaction is negative. X-ray of skull shows nothing abnormal.

Secondary Anæmia, Treated with Liver Diet.

By A. G. MAITLAND-JONES, O.B.E., M.C., M.D. (shown by Dr. K. H. TALLERMAN).

E. J., AGED 1 year.

History.—Child was one of twins ; weight at birth, 3 lb. Was unable to take the breast, and was fed on a cow's milk mixture, and subsequently given Nestlé's milk. Then fed on diluted cow's milk with the addition of bread and butter, milk pudding, gravy, etc.

Is said to have had an illness about seven months ago, characterized by attacks of head retraction and unconsciousness, three attacks of which are said to have occurred in the last fourteen days.

On Admission.—Child, who was rather flabby and unable to sit up properly, was noted to have rather a bossed head, and some flairing of the costal margins, but there was no marked enlargement of the epiphyses. The spleen was just palpable ; the liver was not enlarged, and no glands were felt.

October 31, 1927.—*Blood-count*: Red blood-cells, 3,800,000, hæmoglobin 32 per cent., colour index 0·57; leucocytes 5,420: small lymphocytes 46 per cent., large lymphocytes 10·5 per cent., large hyalines 8·5 per cent. Poikilocytosis and anisocytosis.

Although treated on a satisfactory diet and taking iron and cod-liver oil by the mouth for two weeks, there was no improvement in the blood-count, which was as follows :—

November 14, 1927.—Red blood-cells 3,400,000, hæmoglobin 38 per cent., colour index 0·56. Clinically, however, the child seemed much better.

November 15, 1927.—One ounce of liver soup was given daily. On November 22, 1927, ¾ oz. of raw liver, which in the course of the next week was increased up to 1½ oz. daily, was given.

November 29, 1927.—*Blood-count*: Red blood-cells 3,800,000, hæmoglobin 45 per cent., colour-index 0·6; leucocytes 17,440: polymorphs 45·5 per cent., small lymphocytes 47 per cent., large lymphocytes 1·5 per cent., large hyaline cells 6 per cent. Poikilocytosis and anisocytosis.

Between this date and December 15, during which period he was receiving 1½ oz

daily, the red blood-cells increased by over 1,000,000, and he was discharged with the following blood-count, having gained over 2 lb. in weight:—

January 2, 1928.—Red blood-cells 4,900,000, hæmoglobin 75 per cent., colour index 0·76; leucocytes 8,160: polymorphs 25 per cent., polynuclear eosinophils 2 per cent., small lymphocytes 58·5 per cent., large lymphocytes 9·5 per cent., large hyalines 6 per cent.

Discussion.—Dr. K. H. TALLERMAN said the case showed the remarkable way in which the red cells in the blood increased after giving liver diet; it seemed too definite to be due merely to recovery from rickets under cod-liver oil treatment. Another case was recently under Dr. Maitland-Jones, and for a month was treated with iron. When that case first came in, the red cells were just under three millions. Iron was then omitted, the diet being the same, and liver was substituted for the iron. Within three weeks of giving the liver, the red cells showed nearly five millions. During the same time the hæmoglobin was not increased. Perhaps if the iron had been continued also, the hæmoglobin would have shown a rise parallel with the increase in red cells which seemed brought about under such treatment.

Dr. B. E. SCHLESINGER said that Professor Minot, who introduced the liver treatment, found that it was not successful in secondary anæmia in man, although the original discovery was made in association with secondary anæmia in dogs. He would have felt more convinced that cure of the anæmia in this case had been directly due to liver treatment if the results of adding cod-liver oil to the diet had been watched for a longer period than two weeks before commencing liver treatment. More than two weeks was needed for the full effects of cod-liver oil to become manifest in rickets. He understood that cod-liver oil had been continued in association with liver treatment and it was not inconceivable that improvement of the anæmia was entirely due to the more prolonged use of antirachitic remedies.

Dr. HELEN MACKAY said that in a few cases she had been trying to compare the effects of iron and of liver treatment in premature infants, such as the one shown, in whom secondary anæmia had developed. The period needed to get a response from iron varied. She had seen cases in which, after practically no increase in the hæmoglobin content in the blood during three weeks, a very rapid increase occurred. In some cases, on the other hand, there was an immediate response, the hæmoglobin beginning to rise within a week of beginning treatment with iron. She could not yet make any statement on the relative value of the iron and the liver treatments, but her best comparison was in the case of twins, aged 16 months, treated at the Queen's Hospital for Children. One of the twins came in with 26 per cent. hæmoglobin, and after twenty-two days of liver treatment it had gone up to 49 per cent. hæmoglobin. The other twin in the same ward was given an identical diet except that ordinary minced meat was substituted for the liver and iron and ammonium citrate was given. The hæmoglobin in this case rose from 31 per cent. to 66 per cent. in the same time.

Dr. W. S. C. COPEMAN asked whether this patient presented any of the features of cœliac disease, as the blood-count seemed to suggest it. Since the liver treatment had been originated with adult cases of sprue, and a relationship between the two diseases had been suggested, any report of its use in cases of cœliac disease would be of interest.

Dr. TALLERMAN (in reply) said that in this case there was nothing typical of cœliac disease; the stools were normal and there was no diarrhœa. He had been glad to hear the comparison in the results from iron and liver treatment. Even in the short time mentioned the liver seemed to have a very pronounced effect in increasing the red cell count, and this was also noticeable in the other case he mentioned, which however, was not shown.

Dwarfism.

By A. G. MAITLAND-JONES, O.B.E., M.C., M.D.

W. N., FEMALE, aged 3 years and 10 months.

History.—Full-time child, weight 1 lb. 6 oz. at birth. Artificially fed. Although active and vigorous, has grown very slowly. Teeth appeared, and the child sat up at six months, began to speak at sixteen months, walked at two years and four

months. At eighteen months: weight 6 lb. 13 oz., height 22 in. Now takes ordinary diet.

Family History.—Parents alive. Mother has ? pernicious anæmia. No history of dwarfism in the family. One other child, aged 6, alive and well.

Present Condition.—Weight 9 lb. 12 oz. Height 26 in. Head 15 in. The infant, although so small, has an appearance of age. This is very noticeable in the face, the hands are, in shape, those of an adult, the skin being dry and harsh. The blood-pressure cannot be determined, the urine is normal, the blood-urea is 0·036 grm. per 100 c.c. of blood.

Three Cases of Pneumonia with Deviation of the Heart towards the side of the Lesion in Young Infants.

By A. G. MAITLAND-JONES, O.B.E., M.C., M.D. (shown by Dr. K. H. TALLERMAN).

M. G., AGED 1 month. Admitted to hospital with typical symptoms of pneumonia. History of two days' cough, fever and embarrassed respiration.

February 23, 1928.—Crepitations and rhonchi heard over both lungs.

March 3, 1928.—Slightly diminished air entry and impaired percussion note, right lower lobe.

March 6, 1928.—Radiogram showed the heart and trachea both lying to the right with a definite consolidation of the right upper lobe.

March 8, 1928.—Impaired percussion note, diminished breath sounds, right upper lobe, with crepitations throughout right lung. Area of cardiac dullness not defined. Apex beat appeared to lie below the fourth space beneath the sternum.

March 13, 1928.—Radiogram showed apex of right lung clearing with the heart slightly more normal in position but still well to the right.

March 30, 1928.—No definite physical signs now in chest, and heart appeared in normal position on clinical examination. Radiogram taken at this time showed heart and trachea returned towards the left and the apex of the right lung clear.

Child putting on weight and general condition good.

E. A., aged 4 months. Said to have had bronchitis for three weeks. Three days before admission had a convulsion, and on the day of admission a further convulsion with feverishness and embarrassed respiration.

On admission (February 22, 1928) respiration rapid: rhonchi heard throughout the left lung with harsh breath sounds. Over right lung, breath sounds were high pitched; percussion note was impaired; crepitations and rhonchi were heard throughout all lobes; the area of cardiac dullness could not be defined since there appeared some emphysema over left side of chest, but the heart sounds were heard rather better to the right of the sternum. Spleen palpable.

February 27, 1928.—Radiogram shows heart and trachea displaced to the right and areas of opacity of the right middle and lower lobes.

March 1, 1928.—Respiration becoming normal. Left lung clear except for a few crepitations at the base. Right lung: physical signs remained the same although the percussion note posteriorly was now normal. Heart sounds heard more clearly to the left than previously.

March 6, 1928.—Radiogram shows heart and trachea in approximately the same position, though the lung itself is clearing. The chief area of opacity was well seen on the screen, but is not so obvious in the X-ray film, being overlapped by the heart shadow.

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March 20, 1928.—Percussion note impaired and crepitations present at right base. Heart still appears to the right.

Radiogram shows lung clear and heart returning to, although not yet in normal position.

March 27, 1928.—Crepitation still present at the right base. Heart appears to be normal in position now.

April 17, 1928.—Child seemed very fit. Physical signs at right base unchanged.

Radiogram shows right lung clear, and the heart and trachea now practically normal in position.

L. M., aged 10 months. Three days' history of vomiting and "being off his feed".

October 10, 1927.—On admission infant was very cyanosed with embarrassed respiration and marked stridor. There seemed to be much mucus in the trachea: a throat swab was taken and proved to be negative. Coarse crepitations at both bases and rhonchi throughout both lungs. Heart appeared normal in position. Spleen was palpable.

October 25, 1927.—Dullness and diminished breath sounds right base.

No area of cardiac dullness defined. Heart sounds heard most loudly just medial to right nipple.

Radiogram shows no cardiac shadow in left side of the chest, but an opacity throughout right side with trachea pulled over to the right. The picture was taken under difficulties, oxygen being administered by nasal catheter.

The question of a foreign body in the bronchus was considered at this time.

October 28, 1927.—No change radiologically.

October 31, 1927.—Dullness, bronchial breath sounds and rhonchi throughout right lung, with definitely deficient movement of right side of chest.

November 3, 1927.—Percussion note still impaired. Breath sounds high-pitched over right lower and middle lobes. Heart displaced to right and respiration rate still raised, but temperature now normal.

From now onwards the right lung cleared and the heart gradually returned to the normal position, as shown by physical signs.

November 12, 1927.—Radiogram still shows area of density, probably an unresolved patch of pneumonic consolidation. The heart returning to the normal position.

December 1, 1927.—Radiogram showed unresolved area much improved.

December 17, 1927.—Physical examination showed no physical signs whatever, and the heart appeared normal in position.

Radiogram shows the heart and trachea to be still somewhat displaced in position, and the unresolving area to have increased in size.

December 28, 1927.—Eight days after discharge from hospital to the out-patient department the child had a relapse which was marked with cyanosis, dyspnœa, a croupy cough and much frothy expectoration. Owing to lack of accommodation arrangements were made for his admission to the East London Hospital for Children, where he was placed under the care of Dr. G. Bourne, by whose courtesy I was subsequently permitted to examine the child, and who supplied me with the following notes of the case, with permission to report them.

The percussion note at the right base was impaired: bronchial breath sounds were heard at this area and moist sounds throughout both lungs. The heart sounds were best heard to the right side of sternum. The area of cardiac dullness could not be defined.

December 31, 1927.—The dullness and bronchial breath sounds were spreading throughout the whole of the right lung. Moist sounds were heard at the left lower lobe.

January 3, 1928.—Laryngeal stridor very marked: apparent collapse of whole of right lung and marked cyanosis.

January 6, 1928.—Child much the same, except that the whole of left lung was full of moist sounds.

Child died that evening.

Post-mortem Examination.—The heart was found to be lying almost entirely to the right of middle line. There was a small effusion (? terminal) in the pericardial sac. The left lung was large, full of air, with some purulent bronchitis at the base and a small abscess at the apex of left lower lobe. Patch of pleurisy over left lower lobe. There was consolidation of the middle and upper lobes of the right lung, with complete collapse of the right lower lobe. The trachea was normal.

Discussion.—Dr. B. E. SCHLESINGER said he had seen two cases of the kind, cases of pneumonia caught in the ward after operation during an epidemic of influenzal pneumonia. He thought the sticky copious secretion in this type of pneumonia might have caused the pulmonary collapse which had been present in both instances. It was astonishing how easily mobile an infant's heart could be. He had recently seen an infant four days old whose heart at birth was in the normal position on the left side of the chest. Three days later it was on the right side, stomach and part of the intestines being shown by X-rays to be in the left pleural cavity owing to a diaphragmatic hernia.

It was then noticed that the position of the heart varied according to the size of the stomach, and tended to assume its original normal position several hours after a feed, only to be again pushed over to the right when the stomach was full.

Dr. TALLERMAN (in reply) agreed with Dr. Schlesinger that a thick, sticky secretion might have had something to do with what was found since after blocking of bronchi there was much more likelihood of there being collapse.

Cerebral Abscess.

By HUGH CAIRNS, F.R.C.S.

IVY W., aged 7 years. When I first saw this child there had been a four weeks' history of Jacksonian fits in the left side of the face, headache and vomiting, and the child was drowsy and was very wasted owing to the frequent vomiting. At operation, a bone flap was turned down, and an abscess was found beneath the "face area" of the motor cortex and the adjacent part of the post-central gyrus. The abscess was drained, and the child did well. Though the operation was only done three weeks ago, the wound is practically healed.

As to the cause of the abscess, there is no evidence of infection from the middle ears, nasal sinuses or mastoid processes. The pus from the abscess yielded a pure

culture of *Staphylococcus aureus*. After operation we obtained the history that the child had had an infected cut on one knee, a few weeks before the symptoms of cerebral abscess began. A few days after the operation a pustule appeared on one finger, and it, like the brain abscess, gave a pure culture of *Staphylococcus aureus*. I think the abscess was primarily of metastatic type, and that the infection entered the body through the cut on the knee.

Dr. Evans, looking up cases of cerebral abscess in this hospital, has found that in 10 per cent., no obviously related focus of infection could be discovered at necropsy in other parts of the body. It is not unlikely that many of the abscesses of this group arise by metastatic spread from some insignificant focus of infection, such as the infected cut knee which appears to have been the source of infection in the case of cerebral abscess that I have shown this afternoon.

Section of Obstetrics and Gynæcology.

President—Mr. COMYNS BERKELEY, M.Ch.

[*April* 20, 1928.]

Specimen: Chorion-Carcinoma of Ovary with Torsion of Pedicle in Child aged 11.

Shown by CHARLES D. READ, M.B., Ch.B.

THE incidence of chorion-carcinoma apart from pregnancy is, according to the literature, rare, and the following case is interesting in view of the history and complication (torsion) of the tumour.

The patient, an only child of eleven years, with no previous history of illness, was admitted to Charing Cross Hospital on December 8 last, under the care of Mr. J. Bright Banister.

Apart from her pallor and rather frail build, nothing unusual was noticeable in her general appearance.

The history showed that perfect health had been enjoyed until one month previous to admission, when she began to lose a scanty blood-stained vaginal discharge.

On the third day she was suddenly seized with acute pain across the lower abdomen, followed by vomiting, the pain later settling in the right iliac fossa. For the first time, a tender swelling was then discovered by the child in the lower abdomen.

Examination revealed the presence of a lobulated swelling extending from the pelvis to 2 in. below the umbilical level, and for the greater part to the right of the mid-line.

The tumour was firm, elastic, mobile from side to side, and only slightly tender. By rectal examination the lower extremity of the tumour was easily palpable and no other abnormality in the pelvic organs could be detected.

Operation.—Cœliotomy was performed on December 9 and the ovarian tumour shown was removed, together with the right tube. It was quite freely mobile without adhesions, but a complete torsion of the pedicle was found.

The left ovary and uterus were normal and there was no free fluid in the abdomen.

Pathology.—The tumour, 6 in. in greatest length and 4 in. in breadth, is coarsely lobulated with many tortuous vessels coursing over its surface. The capsule is intact throughout, and beneath it many hæmorrhagic areas are visible.

On section it is firm and elastic, and presents a somewhat variegated appearance, containing several cystic spaces—some filled with clear fluid, others containing blood.

A few areas composed of pale brain-like tissue are visible, but for the greater part the appearance is that of a diffuse blood effusion. (See coloured Plate.)

Histology.—Sections show, in parts, remnants of ovarian stroma with marked extravasation of blood, but in the area shown under the microscope (and in diagram) a typically trophoblastic structure is illustrated, with well-marked plasmodial masses, large multinucleated cells lining a large blood-sinus, and many polygonal cells derived from Langhans' layer (see fig. p. 68). The whole appearance of the syncytium is distinctly chorionic.

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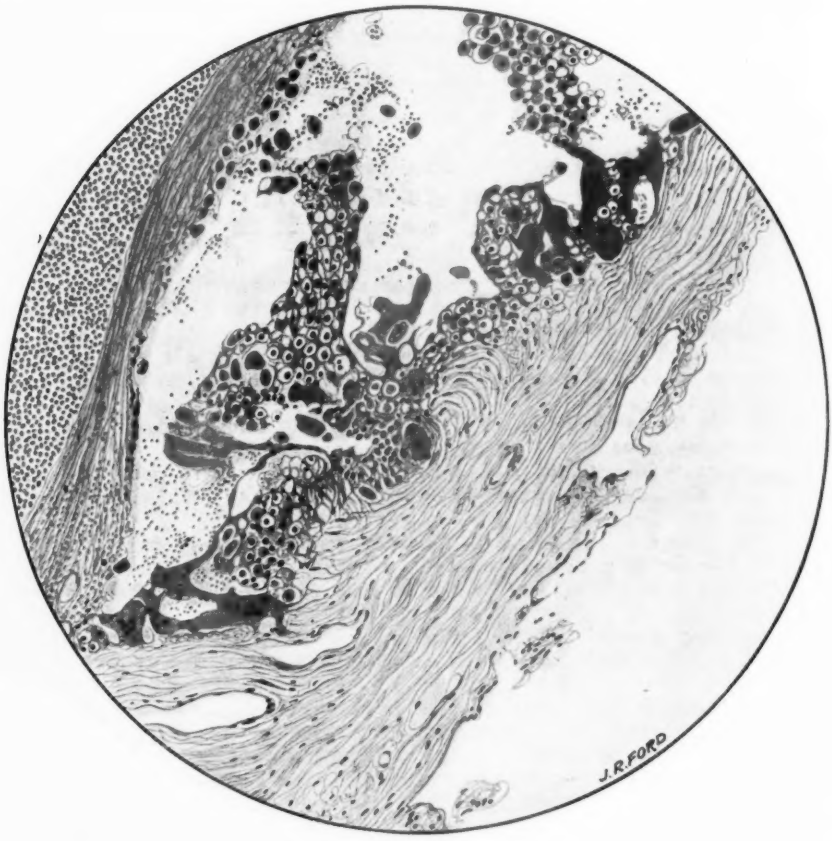

CHORION CARCINOMA OF OVARY.
Showing blood-sinus, syncytium and many polygonal cells derived from Langhans's layer.

The subsequent history of the child is interesting. She made a complete recovery from the operation and left hospital apparently well within three weeks. Nine weeks later a tumour appeared in the parotid gland and one lung was found to be completely dull on percussion. Within a fortnight she died after a series of profuse hæmoptyses.

Unfortunately a permission for post-mortem examination by the family practitioner was not granted.

DISCUSSION.

Chorion-carcinoma occurring apart from pregnancy is rare, but several cases are on record.

In the literature on the subject I find two cases closely resembling the one I have shown to-night.

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Section of tumour, showing variegated appearance and irregular areas of blood extravasation.

READ: Chorion-Carcinoma of Ovary with Torsion of Pedicle in Child aged 11.

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One occurred in a German girl, aged 9 years (Pick), and the other in a girl, aged 16½ years (Michel).

In both these cases the characters of the tumour both macroscopically and microscopically are essentially similar, but in the present case torsion of the pedicle constituted a complication.

To estimate the exact influence of the torsion on the tumour is a mere speculative opinion, but I think to this must be attributed the acute onset of pain and vomiting.

How much it influenced the diffuse hæmorrhagic nature of the growth is an even more debatable subject, as chorion epitheliomatous tumours always show marked blood effusion.

It was only after examination of many sections that anything could be found except necrotic tissue infiltrated with blood.

The interesting features of the case, to my mind, are: (1) Age of patient—11 years. (2) Nature of the tumour—chorion-carcinoma—which, in modern classification, would fall into the groups of embryomata or teratomata. (3) The accompanying torsion of pedicle. (4) The subsequent appearance of lesions in the parotid and lung, which, I think, must be accepted as being secondary deposits derived from the primary ovarian growth.

The treatment of these tumours presents extraordinary difficulties, and one can little more than theorize by advocating early removal. Unfortunately, it happens that this type of tumour manifests itself only at a comparatively late stage, when it is more than probable that metastases have occurred in distant organs.

In those cases occurring apart from pregnancy, it still remains to be proved that removal of the primary growth in any way influences the secondary metastatic deposits as it does in a few recorded cases associated with pregnancy—the metastases shrinking and disappearing, and the patient making a complete recovery.

The cases of Pick and Michel, like mine, both terminated fatally.

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Specimen: Malignant Change in the Corpus Uteri, Demonstrating Metaplasia.

Shown by CHARLES D. READ, M.B., CH.B.

THE specimen shown is a marked example of a well-known pathological change which is fairly generally recognized, though there are some authorities who still hold that the term "metaplasia" is used simply as a cloak for ignorance.

In this case, the patient was a nulliparous woman, aged 34, with a history of excessive loss *per vaginam* during the three preceding periods, and several irregular losses, increasing in severity, over the last month.

An exploratory curettage was performed, and a soft friable mass was found within the uterine cavity—there was free bleeding. The diagnosis of carcinoma corporis uteri was made, and a total hysterectomy and bilateral salpingo-oöphorectomy was immediately performed.

The uterus was a little enlarged, and, on exposing the cavity, two distinct masses were found, one high up in the fundus, the other just above the internal os.

The upper mass is the smaller and has a tufted appearance, somewhat resembling a villous papilloma of the bladder. There is little infiltration of the uterine musculature, and, to the naked eye, little or no ulceration is visible.

70 Read: *Malignant Change in the Corpus Uteri*

The lower growth is larger, more compact, and semipedunculated, and extends well down towards the internal os. Here, again, there is no evidence of ulceration. The cervix appears normal and healthy.

Microscopically, the appearances of the two areas are so dissimilar that it is difficult to believe their common origin.

The upper area shows very definite glandular hyperplasia, with long villous-like processes covered by columnar epithelium. The muscular coat is only slightly invaded. The term papillomatous adenoma would, perhaps, best describe the appearance presented. For the greater part it is definitely innocent in character, but in one place malignant change appears to have supervened. Here, the columnar cells have become stratified and embryonic in character, showing mitosis and loss of basement membrane. This change is most evident in the villous process shown under the microscope and in the diagram.

The lower area presents an entirely different picture. Here the appearance is not unlike that of a squamous epithelioma—the epithelial cells being polygonal, some rounded, and others flattened and arranged in a very definitely stratified manner, producing the appearance of a mosaic with deep processes extending into the underlying tissue. In some parts attempt at cell-nest formation is visible.

In the underlying parts there is a loose œdematous stroma containing many thin-walled vessels.

The interesting features of the specimens are the two apparently completely divorced growths presenting totally dissimilar microscopical pictures.

I am indebted to Mr. J. Bright Banister, both for his permission to publish these cases and for the assistance he has given me.

Professor BLAIR BELL said that since metaplastic changes were a normal feature of the development of the epithelium of the genital passages, it was by no means uncommon to find metaplasia associated with pathological lesions.

It must be remembered that metaplasia from columnar to squamous epithelium was a protective phenomenon: for example, if the vagina remained covered with columnar epithelium after perforation, nearly every woman would die of puerperal sepsis in child-birth, for the vagina would be denuded of epithelium by this performance.

Many years ago he (Professor Blair Bell) had shown that in the case of the imperforate vagina with hæmatocolpos, the epithelium lining the canal was columnar in type in about 60 per cent. of all cases; and recently he had called attention to the occurrence of transitional and squamous epithelium in the body of the uterus containing pus (pyometra). He had seen many other examples of the phenomenon of metaplasia in lesions of the genital tract.

He had not had an opportunity of examining Mr. Read's sections, so would refrain from expressing an opinion about the pathology of the case as illustrated by drawings.

[Professor Blair Bell then gave an epidiascope demonstration of photomicrographs illustrating the occurrence of extensive metaplasia to squamous epithelium in adenocarcinoma of the body of the uterus. He said this was not uncommonly seen in advanced cases, and that although there was cell-nest formation he did not regard the condition as epitheliomatous, or, at any rate, as being specially malignant.]

Eclampsia and Its Treatment: An Experience with Spinal Anæsthesia in One Case.

By R. H. PARAMORE, F.R.C.S.

THE conception that eclampsia is due to a primary failure of the excretory organs (liver and kidneys), the result of ischæmia from too great compression (1909, [23]), seems less impossible than it did six years ago. In 1924 Frank Cook [6] referred to it with some favour. Recently I showed that chronic nephritis in pregnancy rather supports it [27]. To-night I wish to discuss the evidence of

treatment, and to relate a new experience, both of which also point in the same direction.

A knowledge of the cause and mechanism of a pathological process must always be the guide to treatment; inversely, the results of treatment must always check our conception of disease. Up till now the treatment of eclampsia has been empirical; a disease of "theories," eclampsia has been a disease of "treatments." When it was believed that eclampsia resulted from pressure on the renal veins [15], the effect of posture was mentioned—the horizontal position, it was said, prevented eclampsia [11]; and in one case the knee-chest position stopped the convulsions [29]. When it was believed that the ovum, by producing a lethal poison, caused eclampsia, "accouchement forcé" held the field—to be followed by the more reasonable Cæsarean section, with its frequently excellent results. When the impossibility of stating certainly the source of the poison became apparent, and while it was still believed that eclampsia was due to a specific poison of some sort, eliminative measures were pressed, as exemplified by the gastric and colonic lavage of the Dublin school [30], only to be met with the better results of Stroganoff and his treatment with morphia and chloroform, which must tend to lock up secretions and can scarcely be said to be based on the eliminative idea.

Although convulsions do not comprise the essential expression of "eclampsia"—being, according to Mangiagalli, "but an incident of the eclamptic poisoning" [18]—the fits must be controlled, for with their continuance the prognosis becomes more and more grave [7]. Morphia, chloroform, veratrone, blood-letting, emptying the uterus, all have this effect. Experience has shown that morphia and chloroform quieten the nerve-cells and reduce the blood-pressure. Veratrone has been used simply for its effect on the blood-pressure [18]. Morphia and scopolamine, according to Edenhuizer [8], considerably lower the blood-pressure; chloroform does so; even letting out the liquor amnii does so [13]. Delivery, with its usually wonderful effect, could not be supposed to act by directly affecting the nerve-cells. Delivery *must* act, because by delivery the blood-pressure in eclamptics is usually caused to fall, and the blood-flow through the liver and kidneys is thereby facilitated.

The rise of blood-pressure is caused by an increased resistance in the splanchnic capillary areas, due to increased compression of the organs, plus an increased vasoconstriction (of arterioles) in extra-splanchnic parts, due to pressor bodies in the blood, the result of the aberrant metabolism. Many writers think that there is also a constriction of visceral arterioles. Stroganoff believes that the toxin in the blood, the cause of eclampsia, affects the vasomotor centre and especially the blood-vessels, in which it produces "spasm" [33]. Many believe that a spasm of the visceral arterioles—for instance, of the kidney—is the primary pathological change, explaining the oliguria or anuria. But if this were so, letting out the liquor amnii or emptying the uterus would not produce a fall of blood-pressure; and decapsulation of the kidney, post-partum, would have no effect on anuria. Moreover, if we suppose (for the sake of argument) that the capillaries of the kidneys and liver are blocked by compression, the postulation of a renal and hepatic vasoconstriction to explain the raised blood-pressure is unnecessary. Even if pressor bodies in the blood, the presumptive cause of an extra-splanchnic vaso-constriction, tend to produce a constriction of visceral arterioles, there is reason to believe that opposing forces, tending to prevent such vasoconstriction, are in play. When the capillaries of a part are obstructed by compression, the supplying arterioles are reflexly dilated, so that a greater blood-pressure may come to bear on the capillaries in question, and blood may get through. Beyond this is the fundamental fact that the organism can only go on if blood in adequate amount per

unit of time traverses the excretory organs; the whole organism reacts so that blood shall pass through the kidneys and liver adequately. Though we suppose that a constriction of extra-splanchnic arterioles is due to the presence of pressor bodies in the blood, yet these bodies can only be got rid of by hepatic, renal and colonic activity. It thus seems reasonable to suppose that the extra-splanchnic vasoconstriction arises simply, or mainly, to force the blood through the essential viscera—for which a free way through the corresponding arterioles is necessary.

The effect of the remedies used in eclampsia supports this conception. Morphia, chloroform, veratrone, however they affect the vasomotor centre and the arterioles generally, have another effect: they depress the activity of striated muscle, the tone of which is a very important factor in the maintenance of the blood-pressure [27]. The limb muscles act by compressing to some extent the innumerable capillaries [14] permeating their substances; the abdominal wall and diaphragm act in a similar way on the visceral capillaries. The resistance to the blood-flow in the capillaries, produced in this way, plays an active part in the maintenance of the circulation and of the blood-pressure. It is the aortic blood-pressure, in concert with the compression of the capillaries by the tissue cells, which determines the blood-flow through the capillaries, and thus the return of blood to the heart and in part the cardiac response. The activity of the tissue-cells is the ultimate cause of the circulation. The arterioles are secondary conveniences, introduced to facilitate the varying demands. Embryonic and other studies convince one of this.

In eclampsia, morphia, chloroform, veratrone, starvation, all cause a weakness of the muscles—both of the limbs and of the abdomen. Thus, with the drugs mentioned, a greater blood-flow through the viscera is rendered possible; and a greater flow occurs, provided that the capsules of organs (e.g., of the kidney) do not come into play and prevent expansion of the organs from the decompression. If they do, even emptying the uterus is of no avail. The normal emptying of the uterus is presumably without effect on the blood-pressure [31, 32], but after delivery, especially in women who have presented prodromal symptoms of eclampsia, the blood-pressure may rise [12]. For this, one must postulate a constriction of extra-splanchnic arterioles, necessary to overcome a capillary obstruction which persists or develops in visceral parts.

From Stroganoff's experience it would seem that morphia (combined with other narcotics) results in a greater output of urine. "If the fits do not appear for a long time usually all conditions improve; headache is less, the consciousness clears, the quantity of urine increases and the blood-pressure falls" [33]. According to Nicholson [21], both morphia and veratrone are prompt and potent vasodilators and act by causing a relaxation of the renal arterioles. But if the renal arterioles are not constricted—if the oliguria or anuria is due to a partial or complete block of the renal capillaries—the same result occurs if the means employed act by reducing the compression causing the block. Morphia, if it acts at all, seems to act in this way. Although morphia tends to poison or inhibit the activity of renal cells, by reducing the activity of the abdominal wall and thoracic diaphragm and thus allowing a greater flow of blood through the kidneys, it causes an increased outflow of urine—however slight that increase at first may be. Veratrone acts similarly. A poison for striated muscle, if it reduces the blood-pressure it does so at least in part by reducing the compression of the capillaries within the limb muscles and within the large splanchnic area. Chloroform and chloral have the same effect. Emptying the uterus, obviously, greatly affects the physical conditions within the abdomen; it must relieve a too great compression of renal, hepatic and, indeed, of pancreatic and other visceral capillaries. Even letting out the liquor amnii must have this effect, but in lesser degree.

How does venesection act? Stroganoff, referring to its use, says: "Usually . . . the patient became calmer. . . . As a rule, too, I noticed the increase in the amount of urine . . ." [33]—but he only used bleeding as a subsidiary in his treatment of eclampsia. Hingston [12], who gives venesection first place,[1] states that his patients also had morphia. We may believe that venesection in eclampsia relieves the crippled kidneys; but how? Stroganoff thinks it acts by removing from the blood a certain amount of poison. But he only observed good results after moderate bleeding. Moreover, he allows that venesection is followed by "re-entrance of œdema-fluid into vessels" [33]. But the œdema-fluid, it is said, also contains the poison, and perhaps in greater amount than the blood; it is argued that because the prognosis in œdematous eclamptics is less grave than in non-œdematous ones, the poison is locked up in the œdema-fluid [37]. If this be so and œdema-fluid is returned to the blood-stream after bleeding, it is difficult to see how venesection can result in a reduction in the percentage toxicity of the still circulating blood.

The main effect of venesection must be that it reduces the blood-pressure. By affecting in this way the pressure in the cerebral arteries, it tends to prevent more fits. Its action on the kidney is similar. It must either reduce the compression of the organs, by diminishing the strength of the abdominal wall muscles, which presumably could only occur after moderately large bleedings and after some time; or, by reducing the pressure in the renal arteries, it must reduce the turgescence of the kidneys—at least, of the medullary part of these organs. Starvation acts similarly, but is too slow a method for such an end; its value is that by it foodstuffs, which precipitate the fits, are withheld. Even milk should be withheld; no eclamptic's chance of recovery is jeopardized by complete starvation (giving water only). Eclamptics, indeed, become weak [33], but, for this reason, their chance of recovery is greater.

A scrutiny of the treatment of eclampsia supports this view. All the drugs used are poisons and weaken the patient. Especially do they depress the muscles which produce and maintain the intra-abdominal pressure which, in virtue of its effect on the circulation, is such a potent factor in the maintenance of "strength." Just so far as they have this effect they seem to be more efficacious; unfortunately, they have other effects and cannot be pushed too far. For this reason they are often disappointing. Combined with the insistent maintenance of the lateral position, better results with these remedies probably would occur. Placing the patient on her side immediately considerably reduces the intra-abdominal pressure, as observations of the pressure in the rectum show [24]. Unhappily, this position is advised simply to prevent the woman "drowning" in her secretions; thus, she will be pushed on her side only if she is being choked. In the absence of this, morphia, chloroform, veratrone will be given with the patient on her back; and bleeding will be done in this position. Turning the eclamptic into the lateral position should be the first of all interferences; it favours recovery. In some other diseases—for instance, in yellow fever,[2] and after prostatectomy in "stout" men [34]—it seems the same. In eclampsia, although œdema of the lungs may occur early—e.g., after two fits [33]—it is usually a late phenomenon; placing the patient on her side, by affecting the capillary blood-flow within the skull, thorax and abdomen, tends to prevent œdema of the brain, œdema of the lungs, and (equally important) œdema of the abdominal viscera—of the liver and kidneys. Even in the treatment of pre-eclampsia, the lateral position should invariably be used.

[1] In 1922 Hingston abandoned venesection, and from then to 1926 used veratrone instead—with excellent results. (*Indian Med. Journ.*, 1927, lxii, 179.) This has come to my knowledge since I wrote the paper.

[2] See photographs in Noguchi's paper [21], and footnote in my small volume [26].

Reference to post-operative eclampsia must be made. It has been thought that such cases disprove the mechanistic conception of eclampsia. But the evidence rather points the other way. The cases are uncommon. However they and ordinary post-partum eclampsia are to be explained, sudden emptying of the uterus is not regarded as the best treatment for eclampsia. Although many obstetricians still believe that the placenta is the cause of eclampsia, rapid delivery, by whatever method, is now discredited. Indeed, "termination of labour . . . is not even the principal aim" [7]. This attitude, based on a self-contradictory argument, seems inexplicable; if the placenta has suddenly begun to produce a deadly poison, the only thing to do is to get it out as soon as one can. Removing the source of a poison rapidly from the body should not result in disaster; it should save the situation. In eclampsia, if the placenta is the source of the poison, removal does invariably save the situation. The amazing thing is that the advocates of this conception have not urged serum-therapy; although years ago Leith Murray suggested the use of antivenin [20], and recently serum from recovered eclamptics had been tried [19], Young, I believe, persisting in his argument, does not suggest such treatment, and experience has shown that the placenta is not so deadly as has been supposed. Even cases of eclampsia recover without delivery, the pregnancy continuing with a subsequent live birth [5, 35].

An Experience with Spinal Anæsthesia in one Case.

Such conclusions from these specific data, so exactly coinciding with those obtained from contemplating very different phenomena [24, 25], confirmed my opinion. Still, the question arose whether any further test could be tried. The inquiry led to a consideration of the rational treatment of eclampsia. If the pre-eclamptic toxæmia and eclampsia are due to a too great compression of the viscera, then a measure calculated to reduce the activity of the striated muscles of the abdomen should result in an improvement and even cure the patient. What measure better calculated to this end than those already used could be employed? The answer came: "Spinal anæsthesia." It was the result of surgical practice in non-toxæmic cases; observing its effects, I suddenly arrived at the hope that spinal anæsthesia induced in eclampsia would not only benefit the patient, but sustain my conception of the disease.

Spinal anæsthesia has been used before, in toxæmic pregnancy; but, as far as I am aware, only as a means for permitting further procedures. Thus, it has been induced in toxæmic pregnancy for terminating the pregnancy (Cæsarean section); being preferred in such cases because, for one thing, it "adds no element of hepatic toxemia or renal irritation" [3]. On this others also have laid stress [17, 28, 36]. But my idea was to use it in eclampsia as an end in itself, because of the very great reduction in tonicity of the abdominal walls immediately resulting, and which persists for some time. Spinal anæsthesia could not, of course, be expected to remove a poison from the blood, or to check the effect of any such poison in the blood. It could only be expected to have an effect by reducing the compression of the abdominal viscera and, by acting on the limb muscles simultaneously with those of the abdomen, to reduce the blood-pressure—so desirable in eclampsia. By reducing the visceral compression, it would favour the blood-flow through the liver and kidneys, and in this way slowly lead to a restoration of these viscera and thus to an improvement in the state of the blood. The opinion of Arnold [2] supports this deduction; he referred to the markedly "beneficial after-effects on kidney elimination . . . that practically always do result so much more promptly after spinal anæsthesia than after any other."

I had to wait eighteen months for a case—the only one in which I have tried it.

On February 24, 1927, at about midnight, I was informed that a case of eclampsia had been admitted to hospital. I instructed that chloroform should be given and instruments for inducing spinal anæsthesia be prepared. When I arrived I saw the woman lying on her back; she was breathing stertorously; chloroform was being administered. I immediately turned her on her side. As I did so the whole picture altered—the breathing, for one thing, immediately became quieter.

The following is the history. The patient, C. J., a married woman of 23, well developed, had had a child a year ago, and was then seven months pregnant. A month ago her husband was called up to serve in China, and she had to give up her home and come into Rugby, a fact which seemed to have thoroughly upset her. Three weeks previous to admission puffiness of the face and hands had been noticed, and she complained of headaches. On the night of admission she left the supper table, thinking her child was crying, and went upstairs. Presently those below heard the noise of a fall, and found the woman on the floor in a fit. A doctor was sent for and the patient was admitted to hospital at 11.30 p.m. Before admission she had had two fits; her tongue was bitten and two teeth were broken. She had a succession of fits after admission without recovering consciousness. The pulse was 104, full and bounding; respirations, 24; temperature, 98° F. At 12.30 chloroform was administered, and I saw her soon after.

Two ounces of highly albuminous urine were passed through a catheter. The lumbar region was exposed and painted with iodine, and a needle was inserted into the dura; a small quantity of cerebro-spinal fluid, apparently under normal pressure, was allowed to escape; and 1·5 c.c. of a 5 per cent. solution of tropacocaine was injected. The needle was withdrawn, the wound dressed, the chloroform stopped, and instructions given to keep the patient on her (left) side and report if any further fit occurred.

At about 2.30 a.m., with the resident medical officer, I visited the patient. I found her in a semi-drowsy state; she had had no further fit, but had been rather restless, throwing her arms and legs about. In my presence, she vomited and defæcated simultaneously, while still on her side; the visceral action was spontaneous. Her condition seemed much improved. Instructions were given that she should be maintained on her side and that nothing but water should be given to her. At 11 a.m. the report was favourable; the patient had had a quiet night and was quite rational; a further motion had been passed. The blood-pressure was 143 systolic, and 100 diastolic. At 9 p.m. it was 120 systolic, and 80 diastolic; the patient complained of headache. The total amount of urine passed on that day (February 25) was 24 oz. The further progress of the case was uneventful, except that the amount of urine passed did not exceed 30 oz. for the first four days; that the blood-pressure became again raised (on February 28 it was 150 systolic, 110 diastolic); that the fœtal heart-sound was heard on March 1, but was not heard on March 2, and that the patient miscarried on March 4. With the death of the child the amount of urine increased and the blood-pressure fell; after delivery, both conditions improved still more. The urine increased to over 60 oz. on some days, and on March 8 the albumin had disappeared. The patient was discharged on March 19, apparently quite well.

The following questions arise for discussion:—

(1) Was this a case of eclampsia; and if so, was it a slight or severe case?

(2) If it was a case of eclampsia, was the treatment responsible for the result; and if so, was the spinal anæsthesia in any way responsible for that result?

(3) If the spinal anæsthesia was in any way responsible, how did it act?

(4) In view of the effect of spinal anæsthesia on the uterus, was the drug in any way responsible for the fœtal death; and if not, what was the cause of the fœtal death?

(5) In view of the insufficiency of the urinary output during the first four days of convalescence, and the rising blood-pressure, should any part of the treatment, e.g., the spinal anæsthesia, have been repeated? Would that have improved the maternal condition and saved the child's life?

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Discussion.—Dr. T. WATTS EDEN said that he could not agree with the theory of eclampsia which Mr. Paramore had been advocating with such eloquence and sincerity for many years. The theory could not be proved by ingenious argument, and Mr. Paramore would not carry conviction to others unless he could in some way test it by the inductive method. If he could experimentally produce eclampsia by raising intra-abdominal pressure, or if he could show that raised intra-abdominal pressure at times caused eclampsia apart from the pregnant state, he would go far to prove his case. To his (the speaker's) mind the occurrence of post-partum eclampsia might be said to knock the bottom out of the mechanical theory at a blow.

Mr. R. H. PARAMORE (in reply) thanked Dr. Eden for his remarks. Adverse criticism was better than no criticism at all. Eclampsia was such a large subject that it was impossible in one paper to deal with the whole subject; but he assured Dr. Eden that he would not shirk considering the post-partum cases.

He was sorry that the old criticism of Matthews Duncan—that large abdominal tumours caused the same pressure as did at times the pregnant uterus—should have been brought up again. He thought he had already replied adequately to that criticism. The condition of the abdominal wall in pregnancy and its condition in cases of large ovarian cysts were quite different and the pressure in the rectum was not the same.

It was true that a single case could teach little—but it taught something. It was at least an indication—a straw in the stream.

Section of Psychiatry.

President—Dr. R. Langdon Down.

[*March* 29, 1928.]

Joint Meeting with the

Medical Section of the British Psychological Society.

DISCUSSION ON THE ÆTIOLOGY OF ALCOHOLISM.

Dr. Bernard Hart (Section of Psychiatry).

Alcoholism is a subject of immense interest and importance, and its study involves problems which stretch into many spheres of knowledge, medical, psychological, sociological, and political. The domain is so vast, and any attempt to deal with it as a whole would so obviously be futile, that it is necessary carefully to limit and define the field with which the present discussion is concerned.

To begin with, we have to do solely with ætiology and not at all with prophylaxis or therapeutics. We are not directly concerned, moreover, with the effects which alcoholism may produce, whether these be medical, psychological or sociological, but only with the ætiology of the alcoholism itself. The problem of the causation of alcoholic psychoses, for example, lies outside our field. Nevertheless it may be necessary to devote some attention both to this problem and to the various other effects which alcohol exercises upon our physiological, psychological and sociological life, in the hope that we may discover therein information which will throw light upon the factors responsible for the primary alcoholism. Alcoholism we shall define roughly as the continued and excessive ingestion of alcohol, and leave for the time being the important question whether and how far such excessive ingestion is capable of being subdivided into independent types.

The subject of our inquiry may therefore be expressed quite simply as: "Why do certain people take alcohol to excess?" To this question many answers have been returned. Leaving on one side the facile explanations that alcoholism is a "vice" or a "disease," explanations which add nothing to our knowledge and only bring us the comfort of a word, the theories which have been put forward range over a wide variety of factors. Some of these theories emphasize the part played, in one form or another, by heredity. Others, again, attribute primary importance to the mechanism of habit, and the establishment of a vicious circle whereby the consumption of alcohol itself initiates processes which lead to its repetition. Finally there are the theories which seek an answer in psychological conceptions, and endeavour to explain alcoholism as a response to stresses and strains occurring in the mental organism. The psychological theories are of comparatively recent origin, at any rate in any clear-cut form, but at the present day they tend to dominate the field, and to push into the background the factors emphasized by the older views. It would appear, indeed, that these other factors are now in danger of being unduly minimized and neglected.

The question: "Why do certain men take alcohol in excess?" may be coupled with the question: "Why do men take alcohol?" and it may reasonably be anticipated that in the answer to the latter we shall find factors which will at any rate go far to answer the former. Nevertheless any adequate appreciation of this

apparently obvious statement has only been reached in the modern psychological conceptions, and is almost altogether lacking in the theories of the older investigators. For them the fact that men take, and like to take, alcohol, was accepted as obvious and "selbstverständlich," and for them the essence of the problem of alcoholism lay, not in determining why alcohol should tempt, but why the defences against temptation should fail. Hence the emphasis was laid upon hereditary weakness or perversion, or upon the establishment of vicious habits. The modern psychological approach, however, links the two questions together, and holds that the forces which drive men to take alcohol to excess are in some measure at least identical with those which drive men to take alcohol at all. Reasoning along these lines it would seem that the causation of alcoholism must be sought after by inquiring into the effects of alcohol, because somewhere amongst those effects will be found the goals for whose achievement alcohol is taken.

Stockert[1] divides the effects produced by alcohol into two groups: (1) Psychomotor stimulation coupled with an expansive and euphoric affective tone; (2) a dissociative "dulling" process extending to paralysis. This description is neither sufficiently detailed nor complete for our purpose, but it emphasizes the nature of the essential factors involved, and Stockert's two groups may usefully be employed as a basis for further amplification.

The first group includes on the one hand the psychomotor stimulation produced by alcohol, and on the other the euphoric affected tone. How far the psychomotor stimulation is real rather than apparent is a disputed point, but ergographic experiments have shown that small doses of alcohol produce an actual increase in the output of work during a limited period of time. This objective increase is, however, of minor importance in comparison with the illusory increase ascribable to the altered affective tone. The individual believes that his output is much better than it is, and this subjective distortion continues even when the work produced is actually deteriorating. The euphoric affective tone, of which this subjective distortion is but one manifestation, is a factor of far-reaching importance. To it can be ascribed, at any rate in part, the rosy light which alcohol casts over our experience, its efficiency in drowning conflicts, its capacity to relieve us from the demands of reality, and to smooth over pleasantly our failure to adapt to those demands. Trotter has summarized all this in a passage of terse eloquence: "In the tragic conflict between what he has been taught to desire and what he is allowed to get, man has found in alcohol, as he has found in certain other drugs, a sinister but effective peacemaker, a means of securing for however short a time some way out of the prison house of reality back to the Golden Age." The astonishing efficiency of alcohol as a refuge is apparent, moreover, not merely in the way in which it meets an immediate need, but in the continued service it affords to the drinker, so that many of the characteristic symptoms of chronic alcoholism, and of the alcoholic psychoses, themselves give further aid in the escape from reality and internal stress. We may cite here the well-known humour of the drunkard, his almost charming irresponsibility and facility in placing the blame for his shortcomings on wife, employer, or anyone but himself, and those more complex examples of projection which occur in alcoholic paranoia. There are of course other factors involved here than the production of a euphoric affective tone, but the part played by the latter is none the less of fundamental importance. It would be interesting to consider the nature of this euphoric affective tone and the causal processes responsible for it, but

1 F. G. Stockert. "Zur Frage der Disposition zum Alkoholismus Chronicus." *Zeitsch. f. d. gesamt. Neurol. u. Psychiat.*, 1926.

2 W. Trotter. "Sociological Application of the Psychology of Herd Instinct." *Sociological Review*, 1908

this inquiry would take us outside the field with which we are concerned. Possibly the euphoria is only to be explained in chemical terms. Possibly, again, it is only in part directly produced, and in part a secondary result of the removal of inhibitions and the consequent release of inhibited impulses.

Passing on now to Stockert's second group, the "dulling" processes, extending up to actual paralyses of certain functions, we may consider here in the first place the release of inhibitions, a mechanism which underlies so many of the phenomena of alcoholism. There is an increased freedom of expression for all those processes which, in a state of sobriety, are subjected to the braking power of higher controls. We find, therefore, relief from such morbid inhibitions as shyness, but also we find a failure of those inhibitions which control immediate urges in the interest of more remote ends, and hence a type of conduct which is at the mercy of every passing desire. The release of morbid inhibitions certainly promotes sociability, and hence fulfils an aim which unquestionably figures prominently amongst the ends for which alcohol is taken. It is possible that this factor is also conditioned by the release of homosexual forces, on which much stress is laid by psycho-analytical investigators.[1] Psycho-analysts also consider that alcohol tends to destroy sublimations and to aid mental regression, thus bringing into conflict with the personality various impulses previously repressed[2]. Pierce Clarke, although he confirms Juliusburger's statement that amongst these repressed impulses homosexual factors play a prominent part, holds that the regression may proceed to various depths, so that auto-erotic, exhibitionist and other trends may be uncovered[3]. The psycho-analytical school has, indeed, put forward fairly precise theories as to the nature and causation of alcoholism, but in view of the fact that Dr. Edward Glover is contributing later to the discussion, I propose to leave this approach to our problem entirely in his hands. In general it may be said that psychoanalysts accept the view that alcohol is taken in order to achieve a psychological aim, this aim consisting partly in the assuaging of conflict, and partly in the attainment of freedom of expression for repressed forces, and they attempt to formulate in detail the exact factors involved in these various processes.

We commenced this brief review of the effects of alcohol in the hope that amongst those effects would be found the answer to the question: "Why do people take alcohol?" With this limited objective it is clearly not necessary to enumerate all the effects of alcohol, because some are of a character which could not possibly be desired. It may be assumed, for example, that no man takes alcohol in order to acquire cirrhosis of the liver. We have, indeed, already carried the review far enough to see the type of effect which is likely to constitute the aim of the alcoholic. The euphoric affective tone, the submerging of conflict, the cloaking of a reality which cannot be satisfactorily conquered, and the release of impulses which cannot otherwise achieve expression, all these are obviously possible ends. It may be accepted that in the wide range constituted by these various ends will be found the psychological factors responsible for alcoholism, although it is a debatable question how far any particular effect of alcohol is to be regarded as an aim, or how far it is a price to be paid in the achievement of some other end. This question arises, for example, in connexion with the release of homosexual impulses. The psychological aims, however, though undoubtedly of preponderating importance, do not exhaust the whole problem, and there are other factors which require careful consideration.

1 Juliusburger. "Zur Psychoanalyse des Alkoholismus." *Zentralbl. f. Psychoanalyse*, 1912.

2 C. Stanford Read. "Psychopathology of Alcoholism and some so-called Alcoholic Psychoses." *Journ. Mental Science*, 1920.

3 Pierce Clarke. "Some Psychological Aspects of Alcoholism." *New York Med. Journ.*, 1919.

The first of these factors is the "craving," which forms an essential feature of chronic alcoholism. The chief characters of this craving are that it does not arise prior to experience of alcohol, and that it disappears altogether after prolonged abstinence from alcohol, although it can be immediately reinstated in full vigour after a single glass. It does not seem to be susceptible of any satisfactory explanation in psychological terms, and the processes involved are probably best understood by the employment of chemical conceptions. The closely similar phenomenon occurring in morphinism is certainly to be attributed to the formation in abstinence of chemical products which require further doses of morphia to counteract their effects, and it is not improbable that a process of the same kind takes place in alcoholism. This theory has been put forward, indeed, to account for the outbreak of delirium tremens. However this may be, the establishment of a craving is obviously a noteworthy factor in the development of alcoholism, and most investigators who have conducted their researches over a wide range of material are agreed that in certain types of individual it may be sufficient to induce the alcohol habit in the absence of any urges of a "refuge" character. Manual workers, for example, owing to the partly real, though largely illusory, increase in power resulting from alcohol, may come to rely more and more on the drug, until with the advent of abstinence phenomena, craving, and continuous increment of dosage, the full alcoholic habit is established. A similar chain of processes may occur in the convivial drinker.

The various psychological aims, craving, and the mechanism of habit may be said to constitute the factors immediately responsible for the development of alcoholism, the factors which may perhaps be called "exciting causes." The question now arises, however, whether these causes act indifferently on each and every individual, or whether certain individuals are predisposed to their action either by hereditary constitution or previous history. This question takes us into the field to which the earlier authorities devoted their chief attention, and both for them and for a number of later investigators heredity was a factor of predominating importance, although it has been conceived to act in many very different ways. Griesinger held that there was a direct inheritance of alcoholism, Forel [1] that alcohol injures the germ cells, and hence leads to the production of descendants of diminished resisting power, who are therefore more likely to succumb to alcohol. Others, again, maintain that alcoholism is a condition interchangeable with various neuroses and psychoses, and hence likely to appear in any psychopathic stock. It would seem easier to explain the phenomenon here in question, not as a manifestation of simple hereditary transmission, but on the assumption that alcoholism, neuroses, and psychoses, are alternative methods of achieving a refuge and escape from a reality to which adequate adaptation has been found impossible. It is in any case difficult to deny, of course, that all these methods of escape are found to occur more frequently in some stocks than in others, and hence that the existence of a constitutional factor must be accepted.

Archdall Reid [2] has put forward a theory in which the hereditary element in alcoholism is given a precise and specific character. He holds that immunity against alcoholic craving is achieved by a process of breeding under the operation of natural selection, so that races who have used alcohol freely for many generations exhibit comparatively little tendency to alcoholic excess, because the individuals in whom a susceptibility to develop craving is marked have been continuously eliminated, each succeeding generation being therefore bred from the less susceptible individuals of

1 Forel. "Hygiene der Nerven und des Geistes." Stuttgart, 1919.
2 G. Archdall Reid. "Alcoholism, a Study in Heredity." London, 1902.

the preceding generation. The assumption is, of course, made that susceptibility to develop a craving for alcohol is a specific and heritable variation, and one therefore which can be submitted to the action of natural selection. Archdall Reid explains in this way a number of remarkable and interesting facts, the comparative freedom from alcoholic excess of such races as the Italians, in whom alcohol has been universally used for centuries, and the tendency to devastating excess found in savage races who have been newly introduced to alcohol. It must be understood that the immunity in question is an immunity against the craving for alcohol and not against its effects, and that an Italian who does drink to excess is as likely to exhibit the physical and mental results of excess as a member of any other race. Archdall Reid's theory has not been generally accepted, and it is certainly difficult to reconcile it with all the multitudinous facets of the alcoholic problem. Nevertheless many of the facts which he adduces are of a striking character, and have not been satisfactorily explained by any other hypothesis.

Kretschmer [1] approaches the question of heredity in alcoholism from a new angle in his division of chronic alcoholics into two groups distinguished by constitutional characters: (1) a pyknic, cyclothymic type, in which alcoholism develops on the basis of a pliable gregarious disposition. These are the sociable or "stammtisch" alcoholics; (2) schizoid types, difficult and nervous people, who strive to find relief in alcohol from their internal strains and stresses.

This viewpoint of Kretschmer's leads us to the consideration of a problem which we deliberately laid on one side at the beginning of this paper, the question of how far the excessive ingestion of alcohol is a process which can and should be subdivided into several distinct types. Many authors consider this subdivision to be of paramount importance. They distinguish sharply between the taking of alcohol as a beverage and the taking of alcohol as a drug, between the man who drinks because it is a custom, and the man who drinks because he cannot stop. No doubt the distinctions emphasized here indicate the operation of radically different factors, but in practice these factors are found to be inextricably intermingled in one and the same individual, and it is questionable how far it is possible to mark off any sharply defined groups. Another differentiation frequently made is that between the chronic alcoholic and the dipsomaniac, and dipsomania has been held to be a definite nosological entity. It has been supposed to be a manifestation of epilepsy, of psychasthenia, of manic-depressive insanity. Dobnigg and Economo [2], however, conclude that it is not a disease *sui generis*, but a symptom complex which may appear in various types of psychopathic individuals on account of their periodic dysphorias, and this view has much to commend it.

It will be apparent that there is a wide range of opinion as to the relative importance to be assigned to the various factors we have considered as possible contributors to the ætiology of alcoholism. Hereditary constitution, prior psychopathic conditions, conscious and unconscious urges striving to achieve relief from conflict or the release of inhibited processes, habit, and the action of customs and traditions, all of these are very differently estimated or repudiated by different authorities. At one end we have such a statement as that of MacCurdy,[3] "The alcoholic is, before he even touches a drop, an abnormal person." At the other end are those who maintain that alcoholism may arise in a normal man, untroubled by internal conflict or urges towards relief, solely as a result of fortuitous environmental circumstances working upon the mechanism of habit.

1 Kretschmer. "Die Veranlagung zu seelischen Störungen." Berlin, 1924.
2 Dobnigg u. Economo. "Die hereditäre Belastung der Dipsomanen." *Allg. Zeitsch. f. Psychiatrie*, 1920-21.
3 J. T. MacCurdy. "Etiological Factors in the Alcoholic Psychoses." *Brit. Med. Journ.*, 1922.

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Stockert[1], who takes a wide view of the problem, concludes that alcoholism may arise in many different soils and for many different reasons. He divides alcoholics into two broad groups, the convivial drinkers (Stammtischalcoholiken), corresponding to Kretschmer's cyclothymic type, and those who take alcohol as a drug (Trinksuchtigen). The latter group he divides again into the weaklings (Debilen), who drink merely because it is the easiest way of obtaining pleasant sensations, and the psychopaths, asocial repressed personalities, who can only adapt to their environment with the help of alcohol, or who can only in this way substitute day dreams for reality. This classification is clearly of a rough and ready character, and is certainly incomplete, but it serves to indicate the many-sided nature of the problem at issue. We may perhaps conclude that the solution of the problem is not to be found in any single or specific mechanism, but that alcohol may serve one or more of several ends, and that various types of individuals, on account of their hereditary constitution or of their prior history, may be more or less likely to avail themselves of alcohol as a means of securing those ends, and hence to engender processes to which habit will ultimately contribute its part.

In an opening paper, the reading of which is not permitted to exceed twenty minutes, it is clearly impossible to attempt more than a very brief survey of the various views which have been put forward as to the ætiology of alcoholism, and of the factors to which causal significance has been ascribed. Any adequate estimate of the relative merits of these various views, or of the weight to be assigned to each of the many possible factors, would require an investigation far exceeding the limited time available, while to expound at length the tenets of any one school of thought in a sphere where so much difference of opinion exists would seem to be incompatible with the proper functions of an opening paper. It is hoped, however, that the various approaches to our problem will be taken up in more detail by subsequent speakers, and that in this way a foundation will be laid upon which it will be possible to build a satisfactory coördinated view.

Dr. Edward Mapother (Section of Psychiatry).

Dr. Hart has wisely abstained from dividing alcoholics into types and adopted the sounder plan of enumerating all possible factors responsible for drinking and excessive drinking, and suggested that in most cases several factors interact.

Alcoholic excess is far more a problem of social than of individual psychology, and if the time comes when it is of purely historical interest, the change will be due more to the politician than to the psychologist.

It is impossible to avoid all discussion of prophylaxis and treatment if one wishes to reach conclusions which are not merely speculations or generalizations. The only way to assess various factors is to study the results of experiments in which each factor is nationally or individually controlled.

Dr. Hart says that, until recently, the fact that men like to take alcohol was regarded as self explanatory, and that it was left to modern psychology to analyse the effects which motivate ordinary drinking; but I doubt whether we have made any material advance in this matter from the standpoint of psychology for some thousands of years, or much advance from the standpoint of neurology since Hughlings Jackson. Dr. Hart distinguished the psychological effects of alcohol, whose quest actuates the drunkard, as euphoria, submerging of conflict, cloaking of reality—release of impulses otherwise inhibited. Most, if not all of these, are recognized in ancient writings such as the "Bible" and the works of Euripides.

[1] Stockert, loc. cit.

The advance of the new psychology seems to consist largely of the translation of poetry into prose and platitudes into jargon.

The Bacchanalia were the most practical recognition yet devised of the effect of alcohol in removing inhibitions. Dr. Hart in discussing this point refers rightly to the way in which deeper devolutions may accompany the one sought. This was recognized by the Greeks. Professor Gilbert Murray in his notes to the Bacchæ says: "It seems possible that the savage Thracians in the fury of their worship on the mountains when they were possessed by the god" (or, as we should say, "drunk") became wild beasts and actually tore with their teeth and hands any hares, goats, fawns, or the like that they came across."

It has been common knowledge for thousands of years that men drink for one or both of two reasons, viz., to get pleasure or evade displeasure. The distinction is of primary importance though not necessarily clear-cut in every individual. In so far as a drinker is of those who seek something positive he is fairly safe. The dangerous drinkers are those who seek peace and those who take alcohol "to do them good."

The majority of normal drinkers take alcohol because, though it may be bad, it makes them feel good. At least they say so, but the first axiom of the new psychology is that what one asserts with conviction about oneself is probably false.

I believe that "palliative alcoholism" is usually alcoholism of the already sub-normal because it implies acceptance of the situation as irremediable and because, unless courage has been broken by disease, very few external situations justify acceptance of defeat and the quest of anæsthesia rather than an attempt to rectify.

Most alcoholism which seeks negative effects is morbid—of course one cannot invert that statement; there is much morbid alcoholism which is at first positive—i.e., either of the pleasure-seeking or of the "doctor's orders" type—and later hardened by habit.

It is difficult to state concisely the problems of alcoholism. To ask whether the factors inducing men to drink are psychological or physico-chemical is not a true antithesis. Presumably all acquired abnormalities in the nervous system underlying mental disorders are inextricably involved with influences starting in the sensorium and physico-chemical conditions such as constitute the vital efficiency of the neurones themselves and the suitability of their nutrition in relation to their activity. The following questions seem most important:—

Why does alcohol promote euphoria?

What influences encourage or discourage the use of alcohol in quest of euphoria?

Why does alcohol act as a narcotic?

In so far as alcohol is taken as a narcotic to allay discomfort, what are the usual causes of that discomfort?

How often is the discomfort solely and specifically a craving for alcohol?

How often is drunkenness designed to allay grief, remorse, misery or fear?

How often does such a mood appear to be largely independent of experience?

How often does such a mood accompany some particular painful memory or some special current trouble?

The questions why alcoholism causes euphoria, and also why it acts as a narcotic, seem only capable of attack firstly by the neurologist and secondly by the biochemist.

The first sub-question really is, What systems of neurones are directly affected in chemically induced euphoria? One can imagine the possibility of direct evidence; but probably there are technical difficulties. Meanwhile evidence might be got by comparing brains of melancholics and maniacs, exalted and depressed, general paralytics, and euphoric cases of disseminated sclerosis. The second sub-question as to what physico-chemical relations between particular cells and alcohol make the alcohol effective must wait till the cells can be identified.

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An important point would seem to be whether euphoria is a result of (*a*) stimulation, or of (*b*) decontrol, and in the latter necessarily coupled with impairment of a high level.

Dr. Hart, following Stockert, seems to favour the idea of actual stimulation associated with psychomotor facilitation as the cause of euphoria, putting these in a separate class from the reductive processes or dissociative dulling, though the latter include decontrol phenomena, such as relief of shyness. The view of Stockaert and Dr. Hart is the more cheerful, and seems sound, but it is certainly not that which prevails among English scientists.

The Committee appointed in 1921 by the Medical Research Council state in their Report, dated 1923, and entitled "Alcohol: Its action on the Human Organism" (p. 37):—

"Although some of the earlier workers on these lines believed they had found evidence of an initial stimulating effect of alcohol, this appeared in all cases to be of but small extent and duration, and later work throws doubt upon the validity of this interpretation of the evidence, and supports the conclusion that the direct effect of alcohol upon the nervous system is, in all stages and upon all parts of the system, to depress or suspend its functions, that it is in short, from first to last, a narcotic drug."

The exponents of the wholly narcotic view seem to evade the question why exactly euphoria should so predominantly result. It seems an unduly cynical view that decreased lucidity and a veil over reality is almost bound to enhance cheerfulness.

Euphoria certainly does *not* occur in the early phases of other gradually reductive conditions like fatigue or neurasthenia. On the contrary, the results of emotional decontrol in such conditions are usually timidity, liability to tears or irritability.

In the half-drunk, also, a lachrymose or sullen state sometimes from the first replaces the usual jovial one. It is of course possible that the predominance of euphoria as the form of emotional decontrol after alcohol is due to the fact that alcohol is taken by special people or under special conditions, e.g., with a lively expectation of euphoria. It would be interesting to know how far euphoria would follow alcohol taken in ignorance.

The next question is: What influences promote the use of alcohol in quest of of euphoria?

Beyond all doubt, social influences are the most important. There is no need to compare social habits in the days of George IV and George V. The most striking of all evidence regarding causation is found in the change of consumption of alcohol during the ten years 1914 to 1923, inclusive. Since the beginning of the century there had been a steady slow fall in the consumption per head of both beer and spirits. Suddenly within the ten years mentioned the consumption of spirits fell to half the pre-war figure, and that of beer to a little over that ratio.

The figures are enormous. Spirits, 32 million proof gallons in 1914 reduced to 15 million proof gallons in 1923. Beer, 35 million standard barrels in 1914 reduced to 19 million in 1923. Clearly the reduction is very largely in drinking by the relatively sober. If all the national drunkards of 1914 had gone for ever dry they could not have forsworn anything near 17 million gallons of proof spirit, and 16 million barrels of beer.

We can take it that moderate alcoholism is not in the main dependent on individual psychology. It is conditioned by such social factors as tradition and interested boost on the one hand, and by cost and difficulty of getting drink on the other.

Turning now to definitely morbid alcoholism, i.e., alcoholism productive of harm, it is very difficult to be certain about the figures relating to production of mental

disorder. So before discussing those I will refer to figures for two unmistakable physical diseases, viz., alcoholic neuritis and cirrhosis. During the period 1909-1914, each of these diseases was four times as frequent per thousand admissions to King's College Hospital as in the period 1921-1926. This supports the following figures of alcoholic psychosis during the same pre-war and post-war periods: *The ratio of post-war to pre-war certified alcoholic psychosis is rather less than one-third in women, rather more than one-third in men.* I am aware of the personal factor in diagnosis and similar fallacies; I have taken five-year periods before and after from three hospitals, each managed throughout by the same superintendent. The number considered is 5,000 to 6,000 direct admissions in each period, the sexes are equally represented throughout, and there is no change in type of patient or prevalence of other psychosis which might vitiate the figures. They remain equally true whether one takes figures of cases *mainly* due to alcohol (i.e., mostly cases of subacute hallucinosis, Korsakow's psychosis and dementia) or those in which alcohol is treated as *one of several* factors.

There is one possible fallacy, viz., that more cases of alcoholic psychosis are being treated elsewhere without certification. As a result of improved diagnosis in observation wards, and of prejudice against mental hospitals, it might be that cases of good prognosis, which would formerly have been sent on, are being treated to recovery without certification. To some extent this is true. I speak from personal knowledge, as I am Consultant to St. Peter's Hospital, receiving the mental cases for observation from four East End boroughs. The prevalence of psychosis as indicated by figures from asylums would need supplementing by figures from the observation ward, and the frequency of different forms of psychosis would be utterly different in the two sorts of hospital. But while there is no doubt that the sort of retention I mentioned is occurring, it is insufficient seriously to vitiate the statement that a reduction of alcoholism morbidity to one-third of its pre-war prevalence has occurred.

It may be asked, What is the point in all this in relation to ætiology? Just this; human nature has not changed in ten years. From a practical national standpoint it is of vast importance to know that two-thirds of the pre-war morbidity was preventable by judicious handicapping in the contest between boost and cost. I think that the major part of morbid alcoholism and alcoholic morbidity is a result of euphoria-seeking plus habit, that it is not, from any practical standpoint, a result of subtle psychological processes, and that it is as yet impossible to say how far the same is true of the residue of alcoholic excess.

What I have said about the importance of environmental conditions is relevant to Archdall Reid's theory that in races using alcohol freely, sobriety is attained through survival of the sober. As Dr. Hart said, the theory of innate sobriety is at least unproven, but it is probably true that each generation receives as part of external or social inheritance a traditional attitude towards alcoholism that varies in different times and places. Such traditions are part of the unwritten law that makes the people of one country or of one race scattered through the world a nation, and thus temperance or intemperance is a national character, though not innate.

This is very clearly seen among the Jews. At Colney Hatch a large proportion of direct admissions are Jews. During the five-year periods before and after the war, exactly the same reduction was visible as elsewhere, but in each period, and alike for men and women, the proportion of alcoholic psychosis was just half that at asylums which are wholly for Gentiles.

Figures prepared by my colleague Dr. Marian at St. Peter's Hospital show the following facts. Of the total admissions to all wards for mental and bodily disease alike 29 per cent. are Jews. Of admission to the observation wards 27 per cent. are

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Jews, 73 per cent. Gentiles. Of the total cases of psychosis in Gentiles, 13 per cent. are alcoholic, of the cases in Jews $2\frac{2}{3}$ per cent.

These facts cannot be explained by anything but traditional national sobriety. Nothing like relative commonness of other mental disorders among Jews will explain away the finding that of psychosis in Gentiles five times as many are due to drink. The result comes out clearly at St Peter's because at that hospital so large a part of alcoholic psychosis is of acute types due to brief heavy convivial drinking; this is rare in Jews; the insidious psychoses of the solitary drinker are not so specially rare among them.

I think what is true of races is true of families. I know of no evidence indicating clearly transmission of a tendency to alcoholism as an isolated taint where the effect of tradition could be excluded.

That brings us to the question of drinking not for pleasure but for relief of displeasure. I am not impressed by the frequency in alcoholics of any specific craving independent of habit and association of ideas. Alcoholics, mostly, are easily weaned. I have never in an alcoholic seen serious physical results indisputably due to withdrawal of alcohol rather than to poisoning by it. I have never had convincing proof that the traditional giving of alcohol in early delirium tremens is beneficial. I thought the most speculative and probably false item in Dr. Hart's paper was the statement that morphine deprivation symptoms were due to formation in abstinence of products which require further morphia to counteract their effects and that alcohol craving was of the same nature. I presume it is implied that something like antibodies are present, and in absence of the drug produce reverse effects. I know of no evidence of such mechanism. Has anyone ever shown either that injection of the blood of an animal that has been habitually morphinized into another animal, before, with, or after morphine, protected the second, or that the injection of such blood alone produces effects opposite to those of morphine, i.e., like those of deprivation?

It is just possible that, with alcohol, tolerance depends on some protecting lipoids in circulation reducing entry of alcohol to the neurones. If so, the lipoids are more likely to be passively protective than productive of reverse effects and no more likely to produce craving than a mackintosh is to make one pray for rain. All the humoral conceptions of drug tolerance and craving seem to depend too much upon regarding the nervous system as if it were a passive instrument, ignoring its intrinsic adaptability and overlooking fundamental principles of neurology such as contrast and double action of all drugs. Considering the problem of palliative drinking in terms of consciousness, what does the drunkard usually try to allay? I think vague unease and restlessness, or diffuse displeasure. One can only judge the importance of special complexes from cases one has seen, including results of attempts at cure by elucidation and rectification of traumatic situations. The impression left on me is that the significance of these complexes is usually secondary and dependent on congruity with the mood which alcohol is taken to relieve. In other cases this mood or the alcoholism itself has created the situation pleaded in excuse.

As to the idea that homosexuality has any predominant or specific relation to alcoholism, does any experienced person, not committed to universal acceptance of Freudian theory, accept this?

Should we not expect a history that some unmistakable sign of overt homosexuality has occurred in a fair proportion of cases? Should we not expect hallucinations and delusions to refer to the topic with fair frequency? During five years among 2,600 in-patients at the Maudsley there were sixty alcoholics. Only these sixty have been sufficiently recorded to constitute evidence, but they have been quite adequately investigated (if an investigation other than psycho-analysis is

adequate), in every case by observers familiar with Freudian theory, and in a good many by believers in it.

The proportion in which there occurred anything that could without perverse ingenuity be translated as former overt homosexuality, or as reference to the trend, was trivial. Other deviations, such as illicit heterosexuality on the one hand, and impotence on the other, abound in the stories of drunkards. Usually it was impossible to consider alcoholism a reaction to impotence, since it had preceded the latter by many years and was obviously a result.

Dr. EDWARD G. GLOVER (British Psychological Society).

When psycho-pathologists discuss with psychiatrists "the ætiology of alcoholism," both parties must realize clearly the differences in their methods of approach. The psycho-pathologist approaches his problem with a roving commission. His interest in "diseases" is largely secondary, dictated by considerations of convenience in discussion; he prefers to describe as *maladaptations* the various groups of neurotic and associated disorder which come to his notice. He abandons the term "patient" and starts work with a conception of the ego as a psychic organ developed to sample stimuli from the external world and to register inner psychic stimuli (instincts). The alterations effected by the ego in response to these stimuli are described as adaptations, and may take the form of alterations in the individual or in environment. When the individual alteration is detrimental to the interests of the ego it is termed a "maladaptation."

In approaching alcoholism, the psycho-pathologist asks: To what development of instinct life is this a response? Secondly, How does it affect the individual's relation to environment? Thirdly, What individual and environmental factors contribute to the choice of alcoholism as a (mal) adaptation? His problem is primarily one of individual psychology. He is concerned less with statistics of age-incidence than with the reason why any individual should become alcoholic at a particular age. If the alcoholism is uncomplicated he desires to find out why the instinctual difficulties were capable of such direct solution: if the alcoholism is part of a wider symptom complex he wishes to know how the alcoholic response dovetails into other precautions. He then considers certain social factors, to see how far they obscure the individual problem, and the distillate of his combined observations may be regarded as a valid contribution to the psycho-pathology of the condition.

General Psycho-pathological Aspects.—No sooner do we consider the case material than a second difference in approach becomes apparent. The clinician wants to distinguish entities and to show his skill in differential diagnosis. The psycho-pathologist is concerned to discover, for example, what *psychic mechanisms* are common to chronic alcoholism, dipsomania and Korsakow's psychosis, and *then* to discover the mechanisms causing clinical differences.

If we consider clinical manifestations from the point of view of an individual caught between his instinctual urges and the pressure of reality, it becomes apparent that all the primary features of alcoholism represent fundamentally the individual's attempt to extricate himself from an impasse. As far as environment is concerned they are attempts at *flight from reality*, whilst as far as instincts are concerned, this flight coincides with an obvious *increase in phantasy formation*. Clinical differences can then be related to the *completeness* of the withdrawal and to its *periodicity*. The habitual drunkard adopts continuous flight, in contrast to the periodic bouts of the dipsomaniac: neither can compare with the flights in alcoholic hallucinosis or delirium tremens. There is one subjective factor which hinders adequate appreciation of the extent of phantasy activity. The investigator is apt to describe much

of the drunkard's phantasy life by calling him an "inveterate liar" and at the same time to obscure the significance of phantasy in psychotic states by calling it "confabulation." Lying, romancing, boasting, obscene wit, illusion, confabulation, hallucination and delusion, all are results of overcharged phantasy.

But hallucinations and delusions also indicate an attempt to *substitute a new reality for the old*, or at least to *project* into reality the more painful elements of phantasy life. So we must inquire to what extent the alcoholic's overcharge of phantasy life leads to *actual gratification* in *real* life. What is generally called weakness of will, lack of decency, and moral deterioration in the chronic alcoholic can then be described in psycho-pathological terminology as the gratification of previously inhibited impulse. In any case terms such as "weakness of will" combine diagnosis with moral judgment in much the same way that the phrase "obstinate constipation" combines an accurate psycho-physiological diagnosis with an expression of the diagnostician's annoyance.

But it is not enough to say that withdrawal from reality increases or activates phantasy; it activates phantasy corresponding to definite *layers of psychic development*. This element of *regression* can be studied behaviouristically over years in chronic alcoholism, or over a few hours in acute intoxication. The regression of intoxication is plainly heading towards an infantile end, and the ultimate picture is scarcely distinguishable from that of a suckling, who, after an ample feed, might be described as "blind to the world."

The next step in investigation is to catalogue the *instincts or component instincts* gratified in alcoholic regression. Impulses of *aggression* with their accompanying attitudes of hate are freely expressed (irritation, quarrelsomeness, assault). Again, there is accentuation of *sexual impulse* and especially of *component sexual impulses*. Exhibitionism (lack of decency, exposure, obscene wit), scoptophilia (sexual curiosity), sadism (acts of sexual violence), fetichistic activities, manifest expressions of homosexuality, can all obtain representation in alcoholism. On the other hand, in simple intoxication it is easy to find not only direct but *displaced* (and) or *rationalized* expressions of the same impulses (vanity, boasting, anecdotage, exchange of confidences, argumentation, sociability and general "heartiness" towards members of the same sex). Moreover, in alcoholic hallucinosis or paranoia, we see the same impulses in the form of accusations of sexual perversion or delusions of infidelity.

To the *mechanism of projection* involved in the latter instances we will return: meanwhile we may note that it is characteristic of all alcoholic states. The chronic alcoholic shelves responsibility, attributes his drink habits to social or marital difficulties, is suspicious, distrustful and generally "on guard," especially against non-drinkers; the sufferer from delirium tremens may even attack his anxiety hallucinations in environment.

Social Aspects.—Coming to the social aspects of the problem, not all psycho-pathologists are agreed on the ætiology of alcoholism. Some authorities consider that in many instances the unconscious factors can be ignored; they regard social drinking customs, habit, idiosyncrasy, or economic conditions as prime ætiological factors, but the Freudian school holds that external frustration owes its apparent pathogenicity to internal (instinctual) frustrations. Social drinking habits have the same ætiological relation to alcoholism as a pandemic of mild itch would have to acute eczema: moreover, if we examine them closely we can scarcely resist the conclusion that they are socially sanctioned measures of instinctual gratification or defence: witness the joviality of a "stag party" with its scarcely concealed bonds of homosexual interest [1].

The true pioneers of behaviouristic ætiology are the alcoholics themselves, who

are always ready to give rationalistic explanations of their habits. If diagnosticians are to avoid the charge of unconscious collusion, they should be prepared to pay attention to the unconscious significance of *rationalizations*, to say nothing of the language of *symbolism* (compare the homosexual significance of "snake" hallucinations in delirium tremens).

Systematic Psycho-pathology.—The results of psycho-analytical investigation must be considered from three points of view: (*a*) the modification of instinct, (*b*) the Ego's relation to instinct, and (*c*) the relation of instincts and Ego to environment.

(*a*) *The Modification of Instinct.*—A main feature of analyses of alcoholics, drug addicts and manic-depressive insanity is intense preoccupation with *oral* images and phantasies [2]; these are concerned with mouth-breast gratification, or with scenes of frustration accompanied with rage affect. They may be finally expanded into fellatio, cunnilingual, coprophagic or cannibalistic phantasies. The unconcious preoccupations and character traits of the alcoholic indicate either that he has brought about an unobstructed *oral regression* or that his libido has never completely abandoned the oral stage, i.e., that he is *orally fixated*. This fixation is rarely complete even in manic-depressive cases, but we are safe in saying that the alcoholic is *partially fixated*. But since all individuals have oral erotic interests and undergo oral frustration, why are these of special importance in alcoholism? The first reason is that, apart from the gratification of self-preservative urges, oral activities represent the earliest form of libidinal gratification and are associated with the earliest form of aggression and destruction. This particular combination, described as *oral sadism*, is specially accentuated in the alcoholic. The second reason is that excessive frustration in this primitive phase makes the relations to objects (primarily nipple-mother) exceedingly difficult, and *all future relations to objects tend to be coloured by this oral ambivalence.*

The alcoholic then has made a bad start, and whether this is exaggerated by *constitutional* factors or not, he is hampered thenceforward by a tendency to psychic *regression* which produces inertia in development. But matters go from bad to worse. The next stage of libidinal development, the *anal* stage, has more severe frustrations in store. In the alcoholic, *anal erotism* is heightened, again owing to partial fixation, whilst *anal sadism* is correspondingly increased. The result is that when the potential alcoholic reaches the third or infantile genital stage he is faced with a double difficulty: his genital impulses towards objects preserve an archaic sadistic quality, and his reaction to thwarting and disappointment is appropriately severe. His genital libido is impoverished and when the incestuous climax to infantile sexuality is shipwrecked on castration anxiety, the *tendency to regression is fully established.* The ground is thus prepared for miscarriages of sexual instinct in adult life. Most characteristics of alcoholism depend on the *distribution* of this regressional interest. Anal sadistic fixation and regression supply the driving power to *homosexual* impulses [3], which play so large a part in the analysis of alcoholism. Together with other fixations on component sexual instincts, these help us to understand in part the close relation between alcoholism and *perversion* [4]. Finally, the accentuation of genital (castration) anxiety in the alcoholic sheds light on the acute anxieties of alcoholic deliria and psychoses. *One of the dire necessities which drive the individual to alcohol is the need to overcome these excessive charges of castration anxiety.* In the early stages of chronic alcoholism it is partly overcome; the drunkard's boastfulness affords compensation for inner insufficience or impotence (symbolic castration): when however the Ego is temporarily or permanently damaged the anxiety breaks through in massive charges.

(*b*) *The Development of the Ego.*—When the real Ego begins to develop and

become consolidated, a large part of libidinal interest is directed on the self as object (narcissism). Each thwarting produces narcissistic injury, increases ambivalence to objects and makes it more difficult to establish object relations. The most advanced pleasure-producing zone (genital) becomes the centre of narcissistic interest. But this is the zone thwarted in primitive object relations. So between narcissistic fixation, anxiety and regression, the Ego of the alcoholic is overcharged with narcissistic libido. Relations with objects are of course established, but the Ego hardly dares love anyone who cannot be identified with the self. In some cases identification depends upon the object possessing the same genital, incidentally another clue to the homosexual fixations of the alcoholic. When a true object is chosen it is more often than not for revenge, as witness the misery of the alcoholic's marital life.

A second factor in the variety of alcoholic manifestations is therefore *the stage at which the Ego began to lag in development.* Evidence of narcissistic regression is found in the increased use of the defence mechanism of *projection.* The Ego overworks that primitive method of flight from painful excitations which it cannot master, projecting them into a "painful" outer world. This is characteristic of all alcoholic states, although the manifestations vary from vague suspicion to hallucinatory certitude. Some subjects go so far as to pursue these excitations in the outer world and attempt to destroy them (assault, homicide.)

(*c*) *The Ego and Environment.—Many of the guilts and anxieties of alcoholics indicate interference with the function of conscience.* The constant use of projection makes it appear as if their consciences were turned inside out. The alcoholic paranoic detects malignant influences outside himself and rushes to destroy (punish) them; the chronic drunkard feels that everyone else is to blame, especially the virtuous members of his group. Again, the euphoria of alcoholism suggests that relief has been obtained from an intolerable burden, whilst the disregard of social usages indicates temporary suspension of conscience.

Analytic investigation of both normal and alcoholic types shows that what we recognize as conscious conscience is only the peak of a more *primitive conscience,* which operates unconsciously. This archaic conscience (super ego) [5] is a protective organ developed during the gradual wrecking of infantile sexuality: it instigates nside the ego the inhibiting activities at first associated with the parents. Built up when the child's reality sense is weakly developed and when its instincts are exceedingly primitive, this super-ego has primitive characteristics; it demands the repression of incestuous impulse and failing successful repression exacts primitive punishments. Now as the alcoholic tends to remain fixated or to regress to primitive impulses, we must expect to find *either that he has a more severe primitive conscience than normal or that he is unable to cope with the drive of primitive impulse and lands himself in situations of self-punishment.* Both difficulties can be observed in alcoholics. The former is seen in the depressed secret drinker and in more psychotic types whose bout may end in suicide. Up to a point alcohol counters the depression by inducing relative euphoria. This cycle reminds us of the manic-depressive states, and the labile moods of alcoholics are well known. The second mechanism, where inhibition fails and punishments are instituted, is still more obvious. The chronic alcoholic gratifies tendencies which previously may have been foreign to consciousness, and the punitive response elicited from environment varies from moral reprobation to capital punishment. *With the collusion of society the alcoholic has established and for a time reinforced the function of primitive conscience.* The abuse of alcoholism leads inevitably to self-punishment (organic illness, economic failure); it promotes projected self-punishment (aggression towards environment) and this ultimately provokes punishment of the self by environment. In this sense *alcoholism*

is a disastrous attempt to cure abnormalities of primitive conscience. Should the alcoholic attempt to break through primitive inhibitions he risks the integrity of his ego (compare the sequence, latent homosexuality, alcoholism, abstinence, paranoia) [6]; if he maintains an uneasy balance by means of alcohol he is compelled to keep to a course which also ends in physical and mental disintegration.

Why Alcohol ?—To answer this question we must recall (1) the compulsive element in alcoholism; (2) its close connexion with sexual instincts and their components, in particular its compensatory relation to impotence; (3) the fact that sexual instincts are to a large extent capable of displacement; (4) that alcohol is unconsciously identified with bodily secretions and excretions (milk, urine, saliva, semen and blood), and plays a part in unconscious imagery as a medium of sexual assault and as a procreative fluid. It is not too much to say then that *alcohol is chosen as a surrogate of sexuality or, at the least, as a method of short-circuiting sexuality, a method which at the same time releases the pressure of repression and reverses processes of sublimation* [7].

Inter-Relations of Alcoholic States.—To sum up: three main ætiological factors have to be considered in alcoholism. (*a*) The partial fixation of libido at oral or anal sadistic levels of development; the alcoholic maintains ambivalent object relations at the price of alcoholism [8]. (*b*) The constant tendency to regress to a narcissistic Ego organization which automatically sets primitive mechanisms (projection) in action. (*c*) The disorders of primitive conscience which lead to a fruitless exploitation of the same mechanism of projection. We have already noted the resemblance between alcoholic lability and manic-depressive insanity; some forms of alcoholism might be called artificial manic-depressive states. The manic-depressive, however, has a more severe oral fixation of libido, his super-ego is more disordered and his sadism is completely inverted. On the other hand, with a stronger anal-sadistic element, a more primitive narcissistic regression and a disintegrated super-ego the picture becomes one of paranoia; alcoholic paranoia, however, has originally maintained slightly stronger object relations than non-alcoholic. In the next place the dipsomaniac exhibits some of the characteristics of an obsessional neurotic, although alcoholic ceremonial is more apparent in the social drinker. Again, although alcohol brings out many "perverse" sexual tendencies it may itself function as a surrogate of component sexuality. As we know, the essential mechanism of perversion depends on the canonization of one or more component sexual impulses in order that the remainder of infantile sexuality can remain repressed. Alcoholism is in this sense a "pseudo-perversion" [9]. Finally its close relation to psycho-sexual impotence brings us back to the fundamental significance of castration anxiety. We must not forget that the essential problem in depressive states, in obsessional neurosis, fetichism, homosexuality and impotence, is one of infantile guilt and anxiety. Alcohol owes most of its attraction to the fact that it is primarily well adapted to overcome castration anxiety, although in the long run it defeats its own end by bringing about impotence and death (symbolic castration).

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Dr. H. CRICHTON-MILLER.
(British Psychological Society).

In the broadest sense it may be said that alcohol is taken in order to modify the feeling tone. This may be primarily directed to somatic experience, as, for instance, when it is taken to allay fatigue, hunger, or thirst. Or else alcohol may be consumed to modify emotional experience, such as fear or inferiority.

The numerous modifications of feeling tone that result from ingestion of alcohol may, for practical purposes, be referred to two chief mechanisms:—

(1) Interruption in continuity of the functioning of the central nervous system. This interruption may range from the incoördination manifested in one limb to a dissociation amounting to dual personality.

(2) A regressive change whereby the activity of the cortical centres is subordinated to thalamic control, whereby acquired inhibition succumbs to instinctive pressure, and whereby conceptual thinking is submerged by perceptual activity.

It is permissible to suppose that these changes depend on two physical mechanisms, one largely and the other entirely hypothetical.

(*a*) The change in intracranial blood-pressure.

(*b*) The alteration in synaptic functioning, directly occasioned by the alteration in chemical constitution of the cerebro-spinal fluid.

With regard to the first of these, we only know the effect of alcohol in modifying the systemic blood-pressure. As far as I am aware we have no experimental knowledge of the specific effect which it may be presumed to have in altering the relative tension in the intracranial and visceral systems. One might even suppose that the vascular tension is differently affected in cortex and mid-brain.

With reference to the influence of alcohol on synaptic activity, it seems admissible to conceive of this immeasurably delicate mechanism being sensitive to the chemical constitution of the medium in which it functions. Such a hypothesis would serve to explain the range of dissociative phenomena that are met with in alcoholic subjects.

The modification of feeling tone or euphoria thus produced, may be sought under many circumstances. In the first place, any effortless attainment of oblivion must necessarily constitute an alluring retreat from most forms of conflict. The form of dissociation most popular with mankind is that which enables it to forget past follies and future difficulties. Or, to put the same truth in another way, a retreat from conceptual to perceptual ideation is frequently pleasant if not desirable. Thus the introvert finds in alcohol a chemical solvent of those inhibitions that dam up his self-expression. The man with an inferiority sense feels himself 100 per cent. when alcohol has blurred his discriminating capacity. The psychasthenic, with his constitutional intolerance of strain, both physical and emotional, derives from alcohol a temporary rise in the threshold of consciousness.

Similarly, there are two groups whose demand for alcoholic euphoria is based upon physical states—one is the hypopietic and the other the subthyroidic. It is probable that the factor of a low blood-pressure enters into a very high proportion of all cases of alcoholism, especially amongst women, the ill-nourished and the toxic. That alcohol affords satisfaction to many subthyroidics is notorious; presumably, the mechanism is a general vasomotor stimulation. On the other hand, most observers will, I think, agree that hyperthyroidics do not tend to be steady drinkers. Another group that may be mentioned here is the sensorial type. For them the modifications in sensory experience produced by alcohol are sought as are all other such changes. It is in this group that we find those who alternate alcoholism with excessive auto-erotism. One group requires special mention at this point, namely the rebels. The rebel drinker drinks because his father was a rabid temperance reformer, just as the son of the fanatical Calvinist becomes a Roman Catholic and the son of a Tory minister becomes a Fabian. To him the specific appeal of alcohol lies not in its effect on his neurones, but in his father's complexes. This is the group in which gold cures or injections of alkaloids reach the limit of absurdity.

So far I have mentioned some types, all of which tend to become steady drinkers in so far as environment and circumstances are fairly constant. But there are at least three notable groups of periodic or intermittent drinkers.

The first is the epileptic. We know so little of epilepsy that it is difficult to formulate a hypothesis which will cover the dual connexion between alcohol and epilepsy, i.e., the explosive and imperative craving for alcohol as an epileptic equivalent, and the tendency of chronic alcoholism to establish epileptic phenomena that have not hitherto been manifested.

The second group is the dual personality group. In this group profound dissociations occur in which alcoholism may appear to be the determinant of the dissociation, or else alcoholic excess may be a constant feature of one personality.

Finally, there is the manic depressive group—resistant alike to analytical treatment, to forced abstention, to medicinal cures or to affective influences. It is specially to be noted that in this group alcoholic craving may be a feature either of the exalted or of the depressed phase, but, as far as my experience goes, it is never present in both phases.

I have not attempted to be exhaustive, but rather to tabulate broadly. I hope I have said enough to convince some of you who are open to conviction that no simple formula and no single ætiology will cover the pathogenesis of alcoholism, and that in consequence the therapist who deals in a stereotyped treatment is as open to criticism as the magistrate who bases his sentences on the number of previous convictions.

Dr. A. E. A. Carver.

There is one point on which I join issue with Dr. Hart, viz., when he states that the "craving" in a "relapse case" does not arise previous to the retaking of alcohol. In this he follows Dr. F. Hare, but my experience at Caldecote Hall leads me to dispute it.

To give only one example—this week I have readmitted a man who remained teetotal from August last until a fortnight ago. He tells me emphatically that the craving reasserted itself—due to his work "getting on top of him"—before he touched a drop. As the craving is, in my opinion, mainly, if not entirely, psychical, although doubtless, like the emotional states, it has its physical concomitants, one is not surprised if it reappears when stresses, formerly relieved by the drug, recur. The craving is not for alcohol but for its effects.

Dr. Mapother asks whether euphoria follows if alcohol is taken in ignorance? I can definitely answer yes, having made the experiment several times; e.g., in a case of so-called dypsomania, whether the underlying cause be epileptic or manic-depressive, I have wished to give alcohol during the critical period without the patient knowing it, and in such a way that its administration was entirely under my control. This I achieved by adding paraldehyde, the intense pungency of which makes it easy to disguise the flavour of the draught. Euphoria and relief of tension have almost invariably followed the unwitting exhibition of alcohol so given.

Dr. J. A. Hadfield.

We have been presented with all sides of a problem of particular difficulty. But we should not despair of reaching a formula to cover all these views. What little experience I have had of the deep-seated causes of alcoholism has led me to agree with those who regard it fundamentally as an escape from life, a shrinking from responsibilities—a man gets drunk to forget his sorrows.

But in saying this we distinguish between *excessive* convivial drinking and the *compulsive* taking of alcohol as a drug. The former is primarily not a medical but a sociological problem, for "excess" means either excessive from the social point of view, or merely "more than is good for him" (as he might take more meat than is good for him), when it is the *effects* alone which matter. In true "alcoholism" it is not the effects but the morbidity of the causal factors which are of significance and constitute it a disease. Alcoholism proper is a definite psychoneurosis like a compulsive fear or a perversion.

Many factors may produce this tendency to shrink from life and take refuge in alcoholism. One is "*temperamental*," for the alcoholic proper is usually of "highly strung," or "nervous" temperament, particularly unsuited to face the difficulties of life, and therefore succumbs more readily than the robuster type. This introduces the hereditary factor (which has not yet been referred to in this discussion), for our temperaments are for the most part inherited. It is not alcoholism as such that is inherited, but the temperament peculiarly open to it. The *physiological* factor, again, such as low blood-pressure, referred to by Dr. Crichton-Miller, produces the "done" feeling that makes a man feel he must have something to stimulate him. But there seems no doubt that one of the chief causes of the inability to face the responsibilities of life is *psychological*. If the child is brought up in an atmosphere of insecurity, and lacking that sense of protection which is the birthright of every child, he will set out in life with a feeling of apprehension, of anxiety, of lack of confidence to cope with his difficulties. He is arrested in emotional development, in will, in self-confidence: so that when things are too much for him, he is incapable of standing by himself, and finds at least temporary support in alcohol. This arrest is manifest in his whole personality, for alcoholism is never the only symptom in such cases; there is always a neurotic disposition manifesting itself in other ways as well. The general arrest of development, and what Dr. Glover has called "fixation" on an infantile level, may show itself either in a general fear of life, or in other infantile characteristics, such as homosexuality, which gratifies without involving any responsibility.

When the individual cannot face life, he escapes in many ways: one becomes a hysteric, one a pervert, one an obsessional, one an alcoholic. The factors that determine why he should become an alcoholic, and not a hysteric, are probably to be found in a mixture of motives, the most important being that alcohol gives the sense of courage and strength, while at the same time suggesting to him the bliss of sucking in infancy, which was the first way he ever knew of drowning his early sorrows.

Dr. J. D. Rolleston

said that though he was not a professional psychiatrist, he had been interested for many years in alcoholism, particularly in relation to the history of medicine and therapeutics. In a paper read before the Society for the Study of Inebriety[1] he had shown that one of the respects in which alcoholism in ancient Greece and Rome differed from that of modern times was that it was mainly convivial, and that industrial alcoholism did not exist apart from that associated with prostitution. He was surprised that the relation between alcoholism and prostitution had not been discussed, for it involved many interesting ætiological points. The close and world-wide association between alcoholism and prostitution was as well exemplified in classical antiquity as it was to-day. Then, as now, alcohol was consumed in large quantities by prostitutes not only to produce euphoria, but also and mainly to make them forget their degradation. This had been emphasized by many writers on prostitution, especially Parent-Duchatelet,[2] and more recently by Dr. Kurt Schneider, senior physician to the Psychiatric Clinic at Cologne University.[3]

Alcoholism among prostitutes was also doubtless due to some extent to a large proportion of them being mentally abnormal. It should also be remembered that victims of the white slave trade received from their employers a certain percentage of the profits on the drink consumed by their customers and themselves.[4] In dealing with alcoholism of therapeutic origin, Dr. Rolleston said he had for many years been convinced that the unnecessary prescription of alcohol in the treatment of infectious diseases, not only in the acute stage, but also in convalescence, converted abstainers into chronic inebriates. Holding this view during the last two years that he had been in charge of a fever hospital he had reduced the annual consumption of brandy from 2,589 oz. to 25½ oz. without prejudicial effect on the patients.[5]

Dr. W. H. B. Stoddart

said that the essential factor of alcoholism was not the inbibing of large quantities of alcohol but the fact that the patient felt the need of alcohol. Cases had occurred in his practice during the war when the hours of opening of public houses were first restricted. The histories had shown that previously to the restrictions these patients used not to take excessive quantities of alcohol but after the regulations were initiated they got into the habit of entering a public house half-an-hour before closing time and drinking hard to tide them over those hours during which they could not obtain alcohol. Ultimately they developed delirium tremens. It was unlikely that his experience was unique; psychologically, those patients were alcoholic long before they began to inbibe excessive quantities of alcohol.

He disagreed with several items in Dr. Mapother's paper but he had only time to refer to one, namely: his contention that alcohol had no preliminary stimulating effect. Dr. Rivers in his researches on the effect of drugs on fatigue, which included remarkable control experiments, demonstrated such a preliminary stimulating effect quite definitely, even after the ingestion of the equivalent of a tumblerful of brandy. His observers then executed magnificent ergographic tracings, which, of course, very rapidly fell off after taking so large a quantity of alcohol.

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Dr. M. P. LEAHY.

When we ask, "Why does a man drink?" the factors of heredity, temperament and environment have to be considered. Further, it seems reasonable to assume that whatever the apparent cause—physical illness or mental stress—one gives way to drink because the idea "I must have a drink" predominates in consciousness. This is, in my opinion, the determining factor in alcoholism. In short, the ætiology of alcoholism is to be sought in the psychical rather that the physical plane. I make this statement because I have found that by injecting the idea "you do *not* need drink" into the minds of alcoholics, many have refrained from alcohol for periods varying from one to four years, and have again successfully responded to similar treatment. To inject such an idea it is essential to get the mind of the patient into a suggestible state, and this can be done in the majority of cases.

Dr. BRANDER

said that there was no craving for alcohol until it had been once taken. If euphoria resulted, naturally the individual desired to repeat the experience. The euphoria might possibly be due to the presence of alcohol in the cerebro-spinal fluid of the lateral ventricles. Analogous euphoria was found in general paralysis and in disseminated sclerosis, in both of which there were changes around the ventricles. Could the psychological explanation of alcoholic euphoria apply in these diseases also? A body habituated to a substance might experience discomfort in its absence.

Section of Electro-Therapeutics.

President—Sir HENRY GAUVAIN, M.D.

[*April* 20, 1928.]

Observations on the Movements of the Duodenal Contents, with Special Reference to Antiperistalsis and Pyloric Regurgitation.

By R. W. A. SALMOND, M.D., Ch.M., D.M.R.E.

THE duodenum is usually considered as consisting of three parts, the first, or cap, the second, or descending limb, and the third, or ascending limb. The curve or angle formed by these varies very much in different subjects. Developmentally, the cap, like the stomach, arises from the foregut and resembles the stomach rather than the rest of the duodenum both in outline and in behaviour from the radiological, and in its interior, from the anatomical, medical and surgical points of view.

In a paper [1] in conjunction with Dr. C. Bolton, antiperistalsis was described as a normal movement of the duodenum, and its clinical significance discussed. I now record further observations on the movements of the duodenal contents made by means of X-rays. The movements of the small intestine as a whole have long been known but those special to the duodenum have received less attention. This may be because it has been taken for granted that the movements of the duodenum are the same as those of the rest of the small intestine.

Barclay [2], in 1915, said the shape of the cap "suggests an auxiliary sphincter, holding a small supply of food to keep an even flow into the second part of the duodenum." He also stated that if the meal were seen clearly in the duodenum after leaving the cap, the case was one of "duodenal irritation." It should be noted that the observations were made when the meal was a comparatively small quantity of bismuth food made with bread and milk. Case [3] in the same year described the motor behaviour of the normal duodenum, and was then of the opinion that reverse peristalsis was frequently seen. Much of the published work has been on the pathological duodenum; the present paper is intended rather to deal with the normal duodenum, as a clear conception of this should help appreciation of the abnormal.

The following observations were made in opaque meal examinations with special attention to the duodenum and pylorus, no aperient having been taken during the previous thirty-six hours. The meal consisted of four to six ounces of barium sulphate in suspension in a pint of malted milk at body temperature, its passage being watched through the stomach and duodenum with the patient in varying positions. It is fully realized that this is not a natural meal, but some substance opaque to X-rays is necessary for this method of investigation. It is also believed that neither the quantity nor the consistence of such a meal causes irritation of the stomach or duodenum. The movements of the duodenal contents are not only complex, probably more so than in any other part of the alimentary tract, but also they vary in different subjects. In thick persons it was often impossible to make observations, and the best results were obtained with thin ones, when the movements inside the duodenum could be seen in detail. Even in some of these the position of the second and third parts of the duodenum made it impossible to make observations. The following is a summary of what has been observed of the movements of the food in the normal duodenum after the age of infancy:

(1) Peristalsis and antiperistalsis of the cap, usually called contraction of the cap.
(2) Peristalsis and antiperistalsis of the second and third parts.

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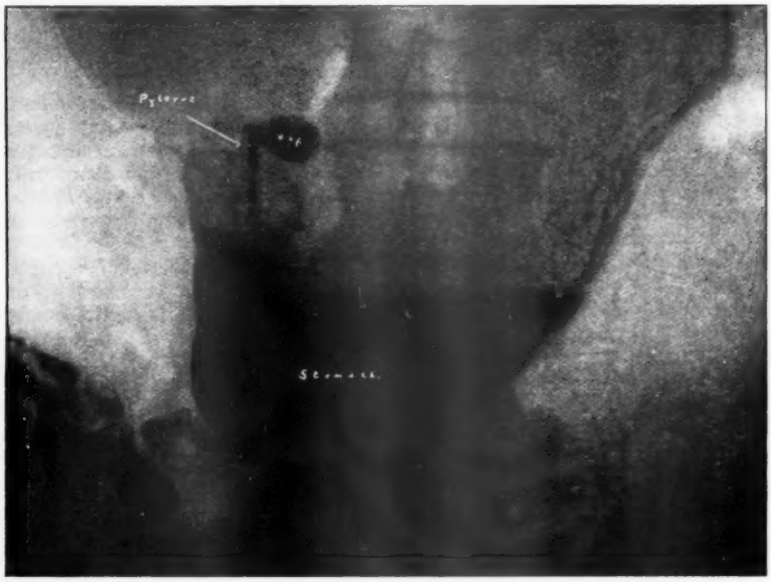
(3) Segmentation or mixing movements of the second and third parts.

(4) Forward movements *en bloc* due to the downward inspiratory movement of the diaphragm, specially affecting the second part.

These have been seen both in normal cases and in the various pathological conditions of the stomach, duodenum, appendix, and gall-bladder.

(1) *The cap*, or first part of the duodenum, appears to act as a reservoir, and to have two functions or phases (*a*) passive and (*b*) active.

(*a*) The passive phase is seen when a gastric peristaltic wave propels the food through the pylorus, fills the cap, and apparently also forces the food on to the second part of the duodenum without any visible contraction on the part of the cap. Another example of this phase is when the filled cap is lying above the pylorus and



suddenly a narrow stream of opaque food flows back into the stomach without any visible contraction on the part of the cap, there being a relaxation of the pylorus and the absence of a gastric peristaltic wave in the pyloric antrum. (See fig.)

(*b*) The active phase. As the cap is filled by gastric peristalsis or, as will be described presently, by antiperistalsis in the other parts of duodenum, it contracts at irregular intervals and the food passes for the most part into the more distal parts of the duodenum or to a much less extent into the stomach. The contraction may expel the whole of the cap contents or only those in the more distal part, the contents in the part nearest the pylorus remaining. No definite relation has been observed between the cap contraction and the gastric peristalsis.

The cap contraction is without any doubt a muscular one on the part of the cap itself. A wave of contraction starts either at or near its base and proceeds forwards

(peristalsis), or towards its distal end and travels backwards (antiperistalsis). This observation is best made in cases with a long drawn-out cap. Whether the cap contraction expels its contents onwards into the other parts of the duodenum or backwards into the stomach depends on different factors, the patency of the pylorus, the relative pressures in the cap and in the stomach at the time, and also whether the contraction wave started at the proximal or distal end.

(2) *Peristalsis and Antiperistalsis in the Second and Third Parts.*—When the food is propelled by the cap or the stomach into the second part it may lie there a few seconds, or it may be at once driven further on by a peristaltic wave. At times this wave drives it onwards only a little, at other times it may propel it into the jejunum, with no delay at the duodeno-jejunal junction, and I believe there is no definite sphincter action at this situation in normal cases. The writer has observed, however, some holding up here in a case of "leather-bottle" stomach, where the function of the pylorus was lost, as if the duodeno-jejunal flexure were trying to act as a substitute. Peristalsis is usually believed to involve a contraction of the gut muscle at one part, with a relaxation further on, and there is nothing special in the peristalsis of these parts of the duodenum as compared with that of the rest of the small intestine. Often, however, before the peristaltic wave or waves can propel the bolus into the jejunum a contraction wave in the opposite direction is set up, and the food mass is driven backwards into a more proximal part of the duodenum, or into the cap, or even into the stomach. The radioscopic appearance of this wave is similar to the peristaltic wave, except that it is in the opposite direction, and if the forward movement of the food is called peristalsis, the backward movement seems entitled to be called antiperistalsis. These waves are quite distinct from segmentation, which does not drive the food one way or the other, but merely breaks it up. If this backward wave has filled the cap, a contraction of the cap is set up sooner or later, and the food, with any that has been expelled from the stomach meanwhile, is again driven forwards into the second or third parts. In this way successive boluses of food are thoroughly mixed together and with the digestive juices. No definite relation has been observed between gastric peristalsis, duodenal peristalsis and antiperistalsis. As might be expected peristaltic waves are usually more frequent than antiperistaltic. Antiperistalsis in the second and third parts may drive the food through the cap and pylorus into stomach, depending on the patency of the pylorus and the relative pressures in the duodenum and the stomach.

It seems, however, that antiperistalsis in the second and third parts of the duodenum is not the only factor in pyloric regurgitation, but that this can be produced, as mentioned above, by the cap in its contractile function, or can occur in its passive phase, the necessary conditions being favourable, namely, relaxation of the pylorus and the intraduodenal pressure higher than the intragastric. Marbaix [4] registers a duodenal pressure of 10 cm. to 15 cm. water, and Payne and Poulton [5] in a normal person found the mean pressure to be 14 cm., with rises to 18 to 20 cm. With a relaxed pylorus these pressures are sufficient to overcome the mean intragastric pressure, which is normally below 10 cm. of water; since, however, with a peristaltic contraction of the stomach the pressure in the pyloric end rises to 20 cm. or 30 cm. of water, or even higher, pyloric regurgitation would be impossible at such a moment. Pyloric regurgitation is now recognized by many authorities as a physiological process, and from these observations, appears to take place in at least three ways, (*a*) antiperistalsis in the second, and perhaps the third and first parts of the duodenum concurrently, (*b*) contraction of the cap alone, and (*c*) a slower trickle through the pylorus when relaxed. In one hundred consecutive cases, specially observed, antiperistalsis in the duodenum was observed in ninety-three, and has

now been seen in over 280 cases. An observer in last month's *Radiology* reported that he had seen it in about 75 per cent. of a small series of cases where it was carefully looked for. The actual regurgitation through the pylorus into the stomach has been seen by the writer in only some twenty-odd cases. Without doubt this small number is due to the very great difficulty of detecting the narrow opaque stream in the pyloric canal, or, if detected, knowing with certainty in which direction it is passing. In stout subjects it is useless to attempt observations on this point.

The movements of the duodenal contents described may help to explain the cause of the symptoms and the results of tests of some of the functional disorders of the stomach and duodenum, and also aid in their treatment. The acidity of the gastric contents appears to be of secondary consideration, for as long as the sphincter acts normally the amount of gastric juice secreted is of little moment. It is therefore important that the exact movements of the duodenal contents and the part played by the pylorus in their regurgitation into the stomach should be made clear.

(3) *Segmentation or mixing movements in second and third parts* are visible as worm-like movements, between the more violent peristalsis and anti-peristalsis, breaking up the mass of food into small particles. Their function seems to be to mix the broken-up food with the digestive juices, and not to move the food onwards or backwards. They are known to occur in the rest of the small intestine and do not appear to have any special characteristics in the duodenum.

(4) *Movement "en bloc."*—When the diaphragm descends on full inspiration there may be seen at times a downward movement of the contents in the whole of the second part as if the pressure of the diaphragm were acting against it and forcing the food forwards into the third part. This is not a movement of the duodenum itself but rather an extraneous factor, but, as it appears sometimes to help the passage of the duodenal contents onwards, it has been included.

In conclusion, these observations have been recorded in the hope that they may be discussed, as the subject is difficult and by no means settled. Duodenal anti-peristalsis is admitted by most to be frequently seen and the crux of the problem would appear to be its significance. Is it normal or is it associated only with such conditions as duodenal stasis, irritation or obstruction or delay even further on? I favour the former for the following reasons: (*a*) the large percentage of cases in which it has been observed, many of them "normal" individuals; (*b*) because it is also seen when giving very small meals, and in the upright, the prone, and the supine positions, and with no delay in the complete emptying of duodenum. Most of these observations could be properly illustrated only by true cinematograph films, and cinematography of these parts is not yet a perfectly safe proposition. I wish to thank my friend, Dr. Charles Bolton, for his great help from the clinical side, and especially for his having been the instigator of many of these investigations.

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Discussion.—Dr. M. Weinbren asked whether Dr. Salmond had seen the "writhing" movement of the duodenum, described by Dr. Jordan, and if so what significance he attached to it.

Dr. Salmond (in reply) said he thought "writhing" movements of the duodenum were usually associated either with an excess of food passed from the stomach into the duodenum which the latter was doing its best to deal with, or with the condition of duodenal stasis which could be due to more than one cause.

Treatment of Incontinence of Urine in Women by Electro-Therapeutic Means.

By L. W. BATHURST, M.D.

INCONTINENCE of urine in women is an extremely common and very distressing condition. It generally occurs in women over 40 years of age, less commonly in those between 30 and 40, and (very) rarely, in my experience, in those under 30, although no doubt circumstances might occur to produce it.

The history generally given is that of a few drops of urine escaping on exertion or laughing, coughing or sneezing, hardly noticeable at first, but later coming more frequently and in increasing quantities, until the stage is reached when there is a gradual dribbling all the time, even in extreme cases when lying in bed.

It will be found on the examination of such a patient that a probe, catheter, or sound passed into the bladder does so with unusual ease and lack of resistance, showing the atrophied condition of the sphincteral musculature.

Besides the misery of continual leaking, there is the necessity for frequent changing of pads to prevent the occurrence of an offensive odour and the risk of excoriation of the external genitals.

As to the ætiology of incontinence, it may be divided into the four following groups:—

(1) Infection by bacilli-coli or other organisms with inflammation affecting the trigone of the bladder.

(2) Stone or growth in the bladder.

(3) Injury to the neck of the bladder and urethra.

(4) No apparent cause.

Obviously, in cases classed under (1) or (2), the cause must be removed before there can be any hope of improvement from any other form of treatment. By far the most common type of injury causing incontinence is that sustained in child-birth, when stretching or bruising has taken place in the musculature of the sphincter of the bladder. Sometimes in these cases there is associated a certain amount of prolapse which, incidentally, seems to benefit considerably by the treatment about to be described. Another cause of incontinence in the "injury" class is dilatation of the urethra by instruments, followed perhaps by insertion of the finger for vesical examination. In my experience these cases do not react so well or so quickly to electrical treatment, and one completely failed to respond, though it had seemed to me a most promising case. The patient was a woman, aged 45, whose urethra had been dilated six weeks previously under an anæsthetic. I am bound to say, however, that it subsequently transpired that there was a strong neurotic element in her case.

In half of the series of cases I am considering the trouble dated definitely from child-birth; in about a quarter of the cases no definite cause could be assigned. I am not suggesting that these proportions are true for all the cases of incontinence, because infections, calculi, etc., have, as a rule, been eliminated before the cases come to me for treatment.

It must be remembered that women have normally a much more slender check on the flow of urine from the bladder than men have. In many women a slight cold or intestinal derangement is sufficient to make them lose control temporarily.

Anatomy.

Mr. Victor Bonney, at a meeting of the Section of Obstetrics and Gynæcology, June 7, 1923, gave a lucid description not only of the anatomy but of the whole process of incontinence. The article was published, accompanied by an anatomical diagram, in the *Journal of Obstetrics and Gynæcology of the British Empire*, 1923, xxx, 358. [Diagram shown.]

In that diagram it is seen that the trigone of the bladder is supported by the pubo-vesical fascia or muscle sheet, which consists almost entirely of unstriped muscle fibre. This is attached anteriorly to the back of the symphysis pubis, and posteriorly to the cervix at its junction with the vagina. Laterally it unites with Mackenroot's ligament on either side. Mr. Bonney states, in reference to incontinence, that "contrary to current opinion, it is not as a rule associated with the more obvious forms of genital displacement, such as cystocele or prolapse of the vault. . . . If, however, the patient in standing, sitting, or squatting position be told to cough hard and suddenly, a very typical displacement will be seen to take place, the urethra and bladder wheeling, as it were, round the subpubic angle, until at the height of the movement the meatus is looking upwards and forwards instead of the normal downwards and forwards, and it is at the moment when this degree of displacement is attained that the spurt of urine occurs. The incontinence evidently depends in some way upon a sudden and abnormal displacement of the urethra and urethro-vesical junction immediately behind the symphysis."

He further states that diurnal incontinence appears to be due to laxity of the front part of the pubo-vesical muscle sheet, so that it yields under sudden pressure and allows the bladder to slip down behind the symphysis pubis and the urethra and peri-urethral wedge "to carry downwards and forwards by wheeling round the subpubic angle." Mr. Bonney says that incontinence occurs only when the anterior part of the pubo-vesical muscle sheet yields, and not when the middle or posterior parts give way, leading in these cases to prolapse of the anterior vaginal wall or vesicocele.

Therefore in the cases of incontinence now under consideration it is obvious that treatment, to be effective, must be directed to restoring function to the anterior portion of this muscle sheet.

In the series of cases which I have treated, and which form the basis of these remarks, I have found prolapse present in only five out of thirty-three cases, or about 15 per cent., and in these we must presume that not only has the anterior portion become functionless but also the middle and posterior to some extent.

In these cases, whatever relief may be given by the wearing of a pessary, it certainly has no effect on the incontinence.

The forms of current which I have employed have been:—

(i) The Bristow coil, which I believe to be effective and is convenient for use in a patient's own house.

(ii) Rhythmic condenser discharges, which I have described in Lewis Jones' "Medical Electricity." In this arrangement the current from the main is passed through a mercury jet interrupter, wired in such a way that a set of condensers are alternately charged direct and then discharged through the primary of a coil and a Lewis Jones rhythmic interrupter, thereby producing an impulse in the secondary which goes through to the patient. This method has an advantage in the case of nervous patients, as the sensory effect, from a clinical point of view, is reduced to a minimum.

(iii) The current I now by preference employ is a combination of the sinusoidal and galvanic current, obtained from a pantostat and passed through a Lewis Jones rhythmic interrupter. In a first treatment, or in the case of a very sensitive patient, no galvanism, or only 1 or 2 ma. are used. Otherwise about 5 to 7 ma. are given.

Mode of Application.

The patient lies on her back on a couch with a saline pad applied to the sacral region. A copper or zinc electrode, wrapped with lint soaked in saline solution, is then inserted into the orifice of the vagina so as to lie immediately beneath the

pubic arch. This electrode is connected to the positive pole, which is said to be the more effective in stimulating unstriped muscle. The current is then turned on gradually and increased in accordance with the patient's feelings, the aim being to give a current of such a strength that the abdominal muscles and those of the thighs and legs may be seen to be contracting and the patient describes a feeling as of being lifted up. The sinusoidal current is increased to the maximum that can comfortably be borne, but the galvanic current is never increased beyond 10 ma.

This current is given for fifteen or twenty minutes at a sitting, and I prefer to give three treatments a week at first, two later and then one.

In my earlier cases I used an intra-urethral electrode, with disappointing results. The current so applied was painful, the pain or discomfort lasting for many hours, after the treatment. Only a weak current, therefore, could be applied, and the pain caused was discouraging to the patient, who was disinclined as a rule to make any attempt at perseverance. This is in marked contrast to the method I have described whereby the patient generally experiences a feeling of exhilaration after each sitting and is only too anxious to continue the treatment.

The number of sittings varies from six to twenty-five or more. Many patients are relieved after two or three treatments, and completely so after six or eight. Others show improvement only after eight or ten treatments.

I therefore make it a practice to warn my patients that although improvement may almost certainly be noted after a few treatments, complete relief may be considerably delayed.

It is difficult to be certain in what way improvement takes place in these cases.

Bearing in mind the anatomy of the female pelvis, with the trigone of the bladder resting on the pubo-vesical fascia consisting mostly of unstriped muscle fibres, it would seem that the loss of control is due to weakness of some of these fibres, and that the good results obtained by electrical treatment are due to stimulation of these fibres and a regaining of power. How far they may be due to stimulation of surrounding voluntary muscles as well, I am not prepared to say. It has never to my knowledge been suggested that these cases are due to faulty innervation, and I do not attribute the results obtained as being due to nerve stimulation.

Taking the cases I have treated since the uniform use of the combined sinusoidal and galvanic currents applied extra-urethrally, I find that of thirty-three consecutive cases, 24, or 72 per cent., could be classified as having been cured, and a number of others were greatly improved.

I have looked into the various electrical methods of treating this distressing condition, as described by Steavenson, Guyon, Bordier, Marques and Rockwell, and in none of them can I find reference to a specific endeavour to improve the tone and function of the anterior portion of the pubo-vesical sheet, and it is only since I have directed my attack specifically to this region that I have obtained the high percentage of successes which I have here described, so that I feel certain that concentration on this particular muscle band is the true objective for incontinence of women.

I can confidently say that of all the patients who come for electrical treatment there are none in whom the results more adequately repay the efforts spent to relieve them. The misery and humiliation which these women suffer can in most cases be relieved to such an extent that a depressed and dreary existence is changed to one of buoyancy and pleasure.

In conclusion, I give as an example the case of a woman, aged 42, who had had two children and had suffered from incontinence since the birth of the first child fifteen years previously. Her condition was a miserable one. She was afraid to laugh or cough, and avoided walking, which was accompanied by an increased

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flow of urine. In her case there was no prolapse of the uterus or vaginal wall. She was depressed in mind and had become fat from want of exercise. In spite of the long period of her disability, she responded quickly to the treatment, which was rather intermittent as she lived a long way from London and came up for short courses at intervals. In all, twenty-five treatments were given, spread over three months, at the end of which time she was able to walk and skip without leakage. A few months later she was hunting, and I have heard on several occasions since that she is well.

Discussion.—Dr. E. P. CUMBERBATCH said that this method seemed to be of considerable value. The customary method, viz., the stimulation of the sphincter by surging faradic or sinusoidal currents, the active electrode being within the urethra, was sometimes successful in cases in which the constrictor muscle was weakened by stretching, as, for example, after parturition. He had under his care a woman, aged 50, who had suffered from incontinence for twenty years, since the birth of her last child. So far the faradic and sinusoidal currents applied by the intra-urethral electrode had failed; he (the speaker) would now try Dr. Bathurst's method.

Cases of urinary incontinence in women and children were sent to him from time to time for electrical treatment. Nocturnal enuresis in children usually responded to it. The immediate object of the treatment in these cases was to procure strong stimulation of the urethra or perineum, and thus to create a more lasting impression on the cerebral centres of impulses from the periphery. The greater number of cases could be cured this way.

Urinary incontinence was sometimes seen in adult patients without a history of injury or signs of organic disease. In the case of an adult girl whose bladder emptied involuntarily when she dismounted from a horse or a bicycle, electrical treatment had been unsuccessful.

The so-called "irritable bladder" in adult females was sometimes accompanied by incontinence. In these cases there was often an unsuspected cystitis. Treatment of the inflammation did not always relieve the incontinence. Intravesical and intra-urethral faradism had been successful in such cases. Last year he (Dr. Cumberbatch) had had a case of this kind, and three applications of pelvic diathermy had effected a cure.

Recently he had had a case of incontinence in a female suffering from the results of myelitis. In this case there was not retention with overflow, but the bladder emptied involuntarily during sleep. Pelvic diathermy was successful in this case also.

Dr. Bathurst had referred to the use of condenser discharges. This method was well worth full investigation, not merely for the treatment of incontinence, but also for paralysis and atony of voluntary and involuntary muscle. The character of the current which accompanied the discharge of condensers of different capacities, the duration of discharge and the rate of rise and fall of the current, should be determined by means of the oscillograph, with the body included in the circuit. Oscillographic records of some currents had already been made, but their character would be altered if the body were introduced into the circuit and the conditions made the same as when the currents were being used for therapeutic purposes. The physiological aspect of the subject had been studied in recent years by Adrian, Lapicque and others, and it was important that the quantitative character of the various electric stimuli should be ascertained if further progress was to be made.

Dr. AGNES SAVILL said that the intra-urethral bipolar electrode toned up the sphincter, but the method was unpleasant for the patient, and it was difficult to maintain the electrode in correct position. For cases with prolapse and cystocele she (Dr. Savill) had found the method of Dr. Zimmern (of Paris) very effective. A well-padded electrode was placed in the vagina, attached to the positive pole; the negative was placed over the pubis, and the galvanic current used for twenty minutes; then with the negative electrode over the sacrum a swaying combined current was given for ten or fifteen minutes, strong enough to cause good muscular contractions of thighs. Probably this toning up of the pelvis circulation and musculature was of great importance. In one case the patient had felt pain during treatment, and had been referred to Mr. Frank Kidd, who, by a cystoscopic examination, had found that there was inflammation of the bladder. It was a good rule to remember that no electrical treatment should cause pain.

Section of Otology.

President—Mr. J. S. Fraser, F.R.C.S.Ed.

[*March* 3, 1928.]

Some Considerations of Vertigo based on Experience.

By Isidore Müller, M.D. (Carlsbad).

Abstract.—Our only direct intimation of the existence of space is the sensation of vertigo, when through our cortical centres we become conscious of the fact that we are surrounded by a "blank" space in which motion is apparent, i.e., the relative condition of our body to the things around us seems to be altered. A precise account of the "nerve-cycle" and of its exact relation to the sympathic and parasympathic—in a word, to the autonomous vegetative—system and to the endocrine system, is at present impossible. We have known since the time of the classic investigations of Flourens, Breuer, Mach, Crum Brown, and their followers—especially Sherrington—that the nature of these things is highly complicated. Simple mechanical theories are useless, even for teaching purposes, as they are more or less fictitious and often incomplete.

The path of nervous action is through the ear, cerebellum, brain stem, cerebrum, medulla oblongata, spinal cord, and autonomous nervous system, to the different parts of the periphery, and there are also mutual connexions with the endocrine glands. Mention is made of the influence of constitution (i.e., inheritance, "the natural endowment"), and also of the *conditional* disposition, temper, training, in a word all external and internal stimuli. Again, the various reflexes, the numerous aggregations of ganglia, indicating a certain autonomy of function, constitute a great problem for future clinical observation. The mechanism of adaptation and compensation by which extensive injury may sometimes be counterbalanced, also enters into the question, as do the varying collateral paths, the synergetic action of different and sometimes very distant organs and so forth. Again, we have to think of the ratio of irritability and fatiguability, a significant factor in causation. Syphilis, arthritis, alcoholism, nicotine-poisoning, anæmia, leukæmia, tumours, and—especially—catarrh, are most important causes.

In dealing with the subject of vertigo and endeavouring to arrive at a clear opinion of one's own—despite the chaotic theories and hypothetical interpretations presented by a great number of workers—and to define the essence and function of the so-called "static organ" or "sixth sense," certain fundamental ideas, e.g., that of space and time, have to be considered, and terms such as "organ of sense," "tonus" and "coördination" must be correctly defined. Finally, the question of inheritance (constitution and disposition) must be taken into consideration.

"Organs of sense" are arrangements of the body able to receive and digest adequate external and internal impulses, which they transfer to certain nerves, of which they are the terminal apparatus, and which conduct the impulses through different stations or relays to the centre, where they become transformed to contents of consciousness.

The idea of "space" differs at different times: the eyes, the organs of touch—kinæsthesia—and the ears, work together to produce it, i.e., it is the psychological result of complex views of the size, form, position, and state of motion of the things around us, produced by the synchytic, harmonious action of these organs of sense. For many years two kinds of space have been distinguished: Space of touch and space of sight, according to the mode of perception. To the determining factors a third should be added—namely, the momentum of motion and muscular action, which is under the control of the "static organ." This third factor has been usually ignored hitherto, because the static organ acts below the threshold of consciousness.

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"Tonus" denotes a specific permanent tension and contractibility of the muscles, which exists in normal cases independently of voluntary innervation. "Coördination" is the result of a harmonious coöperation of groups of muscles for normal sequence, uniformity and expediency of function. This takes place automatically, as a rule; it is seldom under the control of will-power. In this respect all muscles are involuntary and act automatically whether they be smooth or striated. The old distinction of voluntary and involuntary muscles is only admissible in a limited sense, as most muscular actions are automatic and only the first impetus is sometimes under will-power. Mention should here be made of the influence of congenital aptitude, predisposition, constitution, and the coöperation of the autonomous nervous system—sympathetic and parasympathetic—with the endocrine organs. In many ways we are returning to the old idea of neurotrophic influence. This fact and that of the growing knowledge of the interrelation of the endocrines and the autonomous nervous system are of great importance.

Vertigo is the subjective feeling of uneasiness caused by the apparent staggering or rotation of surrounding objects, or of movement of the ground, and the sensation of falling or instability. Purkinje called vertigo a "feeling of disturbance of equilibrium." Hitzig spoke of a mere psychic process.

There are, according to Purkinje's distinction, two different forms of vertigo: the vertigo of touch and the vertigo of sight. We distinguish a lateropulsion and a rotatory vertigo, when the motion is a result of angular acceleration. Both are supposed to occur when our observation is influenced by the static organ (or the reverse), so that there arises the feeling of falling, or of rotation either of ourselves, or of the objects around us. Fictitious motion is the essential characteristic of vertigo.

The "static organ" or "sixth sense" controls the feelings of location and motion of the body and the relation of the body to the perpendicular line and to surrounding objects; i.e., it is the organ of equilibrium.

From the study of phylogenesis it seems probable that during evolution the different sense organs, with their different centres, have become adapted to different impulses. We know fairly well how to differentiate the organs of sense, without knowing at present how to differentiate the centres, though the knowledge that there *are* different centres is based on experimental research and clinical experience, which have resulted in the now well-established theory of localization, and the consequent local diagnosis of brain diseases.

Reference must here be made to Johannes Müller's doctrine of the "specific energy" of sensory nerves, although it has been somewhat affected by later observations which have located the differentiation in the terminal apparatus, or in the centres, or in both combined. The natural processes are more complicated than we used to think, and we must modify our methods of thinking accordingly.

The different organs of sense consist of their terminal apparatus (the receiver), as it were, of the nerve (the transmitter), the different ganglia (intermediate relays), and finally the centre, the cerebral cortex and subcortex, the seat of perception of all internal and external impulses. Physiological experience ends here. The process of forming ideas is a matter for psychology and philosophy.

One of the most important components of the idea of space and time, besides those of sight, touch and kinæsthesia, is the perception of motion in ourselves and in the objects around us. The ideas of space and time together comprise the momentum of motion. The observation and estimation of lapse of time take place automatically through the different parts from the drum, stapes, labyrinth, brain-stem and cerebellum to the cerebral cortex, and vice versa. It can be influenced to a great extent by training, but depends chiefly on the congenital, i.e., constitutional endowment, the different gifts or talents.

The "static organ" is not to be understood as an independent and complete organ of sense such as the eye. No one doubts that he can see with his eyes, but, on the other hand, no one has ever had spontaneous knowledge of a "static organ." Formerly the sole function of the ear was considered to be hearing, but Flourens, Mach, Breuer, Crum Brown and others, have increased our knowledge in this respect. They have attempted to give a mathematical and physical explanation of the different phenomena of equilibrium and vertigo, and this attempt was the first step towards a scientific theory which is, however, still defective and far from complete. The only definite knowledge at which we have arrived is, the ear has two different functions: (1) hearing, and (2) the control of equilibrium, which means, *de facto*, the full control of all movements of the body, i.e., of all muscular actions. We also know that motion is the most significant part of the idea of space and time. There are thus two closely connected points of view: The idea of space and time on the one side and the two functions of the organ of sense on the other. The ear appears to be more fully adapted than any other organ of sense for the comprehension and judgment of time. We know, further, that defects of the ear lead to vertigo, which means loss of control of motion, the most essential principle of space and time. Both are very closely linked together, time being, as it were, the fourth dimension of space. Is there not here a conclusive argument that the ear is the proper instrument for measuring time and space by a harmonious coöperation of both parts, the cochlea and the vestibule? Of course, I am not thinking, for a moment, of an exclusive action of the ear alone, but presume co-operation with the senses of sight, touch and kinæsthesia already mentioned.

The ear seems to me an anatomical, physiological and psychological unit functioning simultaneously and so constituting an appropriate and effective mechanism for the protection of the body and the preservation of life.

I have also other, perhaps more convincing, arguments upon which to found my conclusions. For more than twenty years I have based certain diagnoses and ear treatments on the fact that I could influence vertigo, noises in the ear (and even some forms of insomnia produced by such noises), headache, and deafness, by means of my electromagnetic tuning forks in different keys. I could often "irritate" (i.e., "stimulate") and at other times could tranquillize, by the influence of sound, the acoustic as well as the motor or static function. Further, this effect is produced not only by the direct application of the tuning fork into the meatus but also by the conduction of the sound to the ear through a rubber tube. This has been repeatedly confirmed, and Urbantschitsch and his followers have sometimes created vertigo by conducting the sound of his "harmonika" into the ear. Winkler, in Amsterdam, has lately demonstrated by experimental investigation the close anatomical connexion and physiological interrelation of the whole inner ear (vestibule and cochlea). I am thoroughly convinced by my own long experience that the ear as an organ of sense is a functional unit with different types of reaction resulting from different impulses, and that, precisely for this reason, it is such a susceptible and finely adjusted instrument for the right reception and adequate conduction of centrifugal and centripetal messages. It is well protected, being embedded in the most solid bone of the skull (an excellent example of the economy of Nature). Personally, I do not believe in an entire dissociation of the two parts of the inner ear, but think they are inseparable, two wings of one unit. In spite of this conviction, however, I use for convenience the customary nomenclature.

The constitutional factor to which I have referred seems to depend mainly upon the functions of the endocrine system; I say this with all necessary reservations. There are a great number of diseases of constitutional, i.e., inherited, origin. The products of internal secretion—the hormones—have a great influence on the nervous system

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and are, in turn, influenced by it. For example, the influence on the nervous system of the status thymicolymphaticus and of the secretions of the thyroid, parathyroid and genital glands, is well recognized, and on the other hand, we know from experience the enormous influence brought to bear by the nervous system and the psychic condition—excitement, fatigue, fear, disappointment, sorrow, in short functional nervous affection—on the bodily economy. Again, any one endocrine organ influences another, or all of the others, thus showing the close interrelation not only between the endocrine glands themselves, but also between other organs, and so demonstrating the unity of function of the whole body.

In Carlsbad there are relatively more cases of vertigo than there are anywhere else. When I began to practise there, thirty-five years ago, this was the first thing that struck me, and I lectured to the Medical Society on vertigo during the second season of my practice. I had under observation a street sweeper who suffered from vertigo, sometimes he was unable to stand upright and was thought to be intoxicated. He was, however, a total abstainer. Examination showed a chronic vertigo due to fixation of the stapes; both hearing and equilibrium were affected, and a successful operation completely cured the trouble. Another patient, an American, aged about 50, suffering from chronic Ménière's disease with frequent acute attacks of "seasickness" and vertigo said to arise from "biliousness," had been to Carlsbad. He took the cure but became worse every day and his case was very serious. He never could walk alone, even at his "best" times. Examination showed a chronic catarrh of nose and throat and of both Eustachian tubes. Both drums were thickened and indrawn, hearing was considerably impaired, he heard ear-noises and suffered from headaches, insomnia and great general nervousness. Weber was not lateralized, Rinne was still slightly positive, but Schwabach was noticeably shortened: whisper 1 m.-2 m. Medium-sized bougies passed on both sides only with great difficulty. Treatment: Stretching of both tubes, employing my method of inhalation for nose and throat, and also my method of vibration with my tuning-forks. At first only "deep" forks were bearable, but in the end even the highest were tolerated; hearing 6 m., whisper; the vertigo disappeared and the patient could walk alone and climb the winding stairs to my office without aid. A year later he won a trotting race, and after that I lost sight of him.

Among other cases was that of a prominent lawyer, who had an attack of vertigo (thought by his friends to be an apoplectic seizure) on the railway station just as he was about to leave Carlsbad; and that of a member of the Austrian Parliament. Both were cured by inhalation and vibration carried out according to my method.

One patient had come to Carlsbad after many years' treatment for a condition diagnosed as intestinal and liver trouble, there was nothing of the kind, but there was a chronic catarrh of nose, throat, and Eustachian tubes with middle-ear vertigo. He was treated by the inhalation tubage and vibration methods with satisfactory results.

There are many cases of chronic gastric and intestinal disease—especially colitis, liver trouble and biliousness—combined with vertigo, in which naturally both conditions must be treated if a permanent cure is to be effected. Therefore I use, in every case of ear trouble, with or without vertigo, my two methods of inhalation and vibration respectively. The first hardens the mucous membranes, the second the nerves, and thus they inure patients to fatigue, diminishing sensitiveness and enabling them to resist changes of climate and weather, as well as psychical injuries, and, in a word, fitting them for their work.

In conclusion, it is evident that a complete otological, rhinological and laryngological investigation should be made primarily in every case, and that a thorough local and general clinical examination is the only means of ensuring exact diagnosis and correct treatment.

Discussion.—Dr. J. S. FRASER (President) said that the Section was indebted to Dr. Müller for his lecture. Probably many Members were "unitarians" with regard to the ear. It had always seemed to him hard to believe that the two functions of the ear—hearing and balancing—were so completely separated as appeared from the books.

Mr. SYDNEY SCOTT said that Dr. Müller had alluded to "visual vertigo," and he (the speaker) would like to refer to the forced movements due to visual influences during flying. Some aviators experienced vertigo which was not vestibular in origin, for instance, when looking towards the tip of the upper wing when banking. On shutting the eyes the feeling of vertigo vanished. Also when in a spin, some pilots were affected by definite forced movements, if they looked over and saw the earth apparently whirling round; such forced movements ceased on closing the eyes or fixing the eyes on a relatively stable point, otherwise the pilot might be unable to rectify the machine. Dr. Müller had stressed the frequent correlation of vertigo with post-nasal catarrh, and he (Mr. Scott) appreciated the importance of attention to the Eustachian tubes. No doubt many patients visited Carlsbad before the cause of their vertigo had been diagnosed. When Dr. Müller saw them he found the condition due to Eustachian inefficiency which upset the balance of the labyrinths.

Reference had also been made to the stimulus of the cochlear and vestibular nerves, by means of tuning forks. It was in our experience that certain people would exclaim, "That makes me giddy," when the tuning fork was placed on the mastoid. He (Mr. Scott) assumed that the vibrations of the fork stimulated the vestibular neuro-epithelium. Some deaf-mutes retained their vestibular functions. He recalled the case of a boy, who exhibited stigmata of congenital syphilis, and was deaf in one ear. He found that the vestibular tests on the deaf side reacted normally, and on the other side, where the cochlear was normal, the tests showed that all vestibular functions had disappeared. He assumed that in that case the congenital syphilis had picked out the cochlear nerve on one side and the vestibular nerve on the other. In other deaf-mutes the vestibular as well as the cochlear nerves were defunct, hence the different results experienced with tests in deaf-mutes. Dr. Müller had said that disease of the cerebellum could cause vertigo, but he believed it had been shown that there could be cerebellar disease without vertigo, so long as the vestibular nerves were unaffected. Many patients who had vertigo were wrongly told that it was due to liver trouble, or to some other organic disturbances.

Ophthalmic surgeons admitted that eye-troubles seldom caused true vertigo, though investigation might reveal abnormal conditions as astigmatism, etc. In many cases of recurrent vertigo there were two factors, disturbances in the ear being the essential one, and the other variable.

Dr. Müller had mentioned colitis, sorrow, fatigue, worry, etc., as factors. It was right that patients should be thoroughly examined, and all the possible contributory influences investigated. Even normal patients varied in their degree of sensitiveness to stimulation; some could be rotated in a chair and yet walk straight, because of their great powers of control, whereas others have been sent into convulsions. We must also bear in mind that some patients who complained of vertigo were very susceptible to the power of suggestion.

Dr. DAN MCKENZIE said he had enjoyed an interesting paper on an interesting subject, which, though it had been discussed for many years, was not yet exhausted.

He agreed with the statement that vertigo concerned a great deal more of the nervous system than that directly concerned with the ear; it was one of those sensations which commanded the attention of the whole organism, like tooth-ache or other overwhelming pain; and the mental concentration on it added to the agony. Vertigo could therefore be looked upon as the pain of the vestibular nerve.

He agreed that the two sides of the internal ear might be regarded as physiologically connected. The psychological allusions to space and time were suggestive. One important point concerned rhythm; the influence of that on the economy was a very interesting one; it certainly dominated more of the vital processes than was often imagined. The effect of rhythm in dancing, even in these days of syncopation, and in marching, was very marked, and it was well known that fatigued soldiers marched better under the stimulus of rhythmic music. One would expect, as Dr. Müller had indicated in his paper, and as Mr. Sydney Scott had said some time ago, that the two end-organs of the labyrinth would be simultaneously affected, and that vertigo as a symptom of labyrinth irritation might accompany almost any

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disease of the ear. The juxtaposition of the two organs anatomically was not a mere accident, but was necessary for the functions which were carried out together.

Sir WILLIAM MILLIGAN said that when confronted with a case of vertigo he first thought of possible local causes, then of possible general causes, and, finally, of possible central causes. A large number of cases of vertigo were due to a local cause, it might be in the tympanum, in the Eustachian tube, in the posterior end of the middle-ear cleft or as a result of inflammatory lesions in the middle ear. Many cases were treated successfully by opening up the Eustachian tube, though sometimes that was very difficult. Catheterization and politerization sometimes failed, and in obstinate cases he had sometimes made an incision in the membrane and passed a fine silver bougie from above downwards, instead of in the usual direction, from below upwards. That only applied to cases in which it was impossible to get the equilibrium restored between the outside air and the tympanum. The mechanism of the internal ear was so delicate that the slightest alteration of poise determined whether or not vertigo would occur. It was always best to try to eliminate simple causes first; only passing to more obscure ones if simple causes could not be found.

As to general causes, he thought many cases were due to auto-intoxication. Sometimes by altering the splanchnic tension, or a congestive condition of liver, or an intestinal condition, the vertigo could be made to disappear at once. By the simple measure of giving a little calomel at night and salts in the morning the vertigo was frequently stopped.

The central causes presented a more difficult problem, as he always had in mind the possibility of psychic and subconscious effects, and sufficient was not known about the circulation in the brain to enable confident analysis of those cases to be made. In many cases, however, the condition was transitory and due to a temporary emotional disturbance, states resulting either in a stagnation of the cerebral circulation or in anæmia of the brain. Frequently when a patient blushed vigorously he at the same time felt dizzy. If a man were given alcohol he flushed and felt sensations in his ear and experienced some measure of dizziness; he might even show a tendency to fall, showing how easily the equilibration could be disturbed. The same was true of shock and fright. On receiving a sudden fright the mental attitude was tense and there was a momentary halt in the circulation, with its result on the internal ear.

He did not dissociate the cochlear from the vestibular organ, though the latter was of the greater antiquity, which might have something to do with the difference in their response to sensations.

There was a type of vertigo in which it was very important to consider the blood-pressure and that of the cerebro-spinal fluid and their effects on the ear. Sometimes cases with very low blood-pressure had extreme vertigo; also some cases with very high blood-pressure had it. Such people should have general treatment. There was a type in which the end-organ was so damaged as a result of a pathological condition that, for practical purposes, it had become useless, yet it did not cease to act.

When ordinary local and general treatments failed to give relief, there was need for something to be done to the vestibular portion of the labyrinth. After operations on the semicircular system it was remarkable how extirpation of the disease found cleared up the vertigo, thus showing that it was a peripheral, not a central lesion. Still, in some cases there was probably a central end-effect, though he did not know how a diagnosis of this was to be made.

In dealing with this subject it was very important to eliminate auto-suggestion, though how far relief of that was likely to relieve the vertigo he was not prepared to say. Each case required a good deal of sifting, but many were amenable to ordinary methods of treatment.

Sir JAMES DUNDAS-GRANT said he agreed that in any given case of vertigo the local causes in the middle and the internal ear should first be investigated. The condition of the Eustachian tube would often give the key to the situation. He had shown at a meeting of this Section a man who, as soon as he pressed his tragus, fell down; he had fixation of the ossicles (malleus and incus). As those ossicles were of no use to the patient he (the speaker) had removed them, and the symptom disappeared. He thought the stapes was fixed by the other ossicles, that the pressure was communicated to the membrane of the fenestra rotunda, and that there was no "safety-valve" action on the part of the stapes. He had described many cases of vertigo due to cholesteatoma in the attic, in which removal of the ossicles had allowed an exit and so cured the vertigo.

With regard to the internal ear there arose the delicate question of otolith disease. The profession had been charmed by the descriptions given by the late Professor Magnus and now disparaged by Dr. de Kleyn. Cases of typical otolith disease have been described in which a cerebellar tumour was found. He himself had been misled by concentrating attention on the saccule instead of on the ponto-cerebellar angle. Disease at that spot, if recognized early, could be removed. If mistaken for saccule disease, it might be left until too late. What could be called the "vertigo" centre had been localized by Mills, in America; the late Sir Victor Horsley had accepted it. It was located in the posterior part of the two superior temporo-sphenoidal convolutions. Bonnier, on the other hand, placed it in the ascending parietal. A curious confirmation of this view came to notice recently. A colleague who had a typical "Ménière" attack fell down, and for some days thereafter had weakness in the fingers of the opposite side. This suggested that the equilibration sensory centre was near the hand centre.

Vertigo might arise from irritation, and also from destruction of the labyrinth on one side; in which case it might result from over-action of the opposite labyrinth. He (the speaker) had tested the action of quinine in small doses in checking vertigo, and in cases of unilateral elimination of the labyrinth he found, by cold-air tests, that the quinine, which quieted the vertigo, seemed to do so by lowering the sensitiveness on the sound side. Eventually compensation took place.

One element which was liable to be overlooked was the effect of anæmia; in some cases bromides were given when iron and chloride of ammonium were really indicated.

With regard to the influence of pressure in causing vertigo, he had shown a case in which the stapes had dropped out and the membrane of the fenestra ovalis was exposed. On using the "fistula" test very little vertigo was caused by compression but the slightest rarefaction produced such disturbance that the patient almost fell off the chair. Mr. Lake thought many cases of vertigo were due to diminished blood-pressure, and so he treated them with ernuting. Ammoniated extract of ergot was useful in such cases.

Mr. A. R. TWEEDIE said that with regard to the association between the auditory and vestibular nerves, in his original description of the work of Magnus and de Kleyn, he (the speaker) had mentioned that Winkler drew attention to this relation, as evidenced by the effect of music, e.g., on dancing and marching. He asked Dr. Müller how long treatment took to bring about a cure.

[Dr. MÜLLER: It is difficult to answer that because the cases differ so much; some require no more than a dozen applications; others take six months.]

He (Mr. Tweedie) had come to the conclusion that vertigo was a result of a conspiracy of causes. There was usually both an asymmetrical function of hearing and an asymmetrical response to the tests of the peripheral vestibular organ, whilst, in addition, as evidenced by the galvanic tests, there was generally a hypo-sensitive condition of the vestibular nerves. The other factors were often a local subacute exacerbation in connexion with some form of naso-pharyngitis, altered blood-pressure—excess of alcohol or tobacco—some form of dyspepsia and "age." In his experience these cases tended to get better, or to become accommodated in some one to five years' time. With regard to treatment, this should be directed chiefly towards improvement of the general condition.

Of course it was understood that they were not talking of cases of active suppurative disease.

Dr. MÜLLER (in reply) said his purpose had been to create a discussion, as he felt that such a Society as this should influence future experiments on the subject. Research workers must realize that this was a very complex subject, not to be solved by mere animal experimentation. Clinical experience and a careful local examination must enter into it.

With regard to Mr. Scott's remarks, prospective aeroplane pilots were now scrupulously examined in this respect, since, if anything was wrong with their ears, they could not be efficient pilots. He (Dr. Müller) regarded the labyrinth as the place of control, i.e., the place to which messages were sent. When a man had astigmatism the messages received were false ones, and he fell or had vertigo in consequence. A wrong message meant a wrong directional force. If the controlling organ was very potent it might correct the false message. It was necessary to find what was the ratio between irritability and fatiguability. He would again emphasize the importance of a general investigation in all cases;

he himself always tested the blood and the condition of the kidneys. A patient had been sent to him because of vertigo and deafness. She had been treated by a specialist in Vienna, but without success. He (Dr. Müller) found that she was suffering from severe kidney trouble. This was cured by the Carlsbad waters, and it was unnecessary to do anything to the ears.

Case of Traumatic Destruction of Labyrinth on the Left Side with Hysterical Deafness and Chronic Suppurative Otitis Media on the Right Side.

By SOMERVILLE HASTINGS, M.S.

History.—PATIENT, male, aged 34. Ten years ago fractured skull (depression behind left mastoid), with immediate and complete deafness on left side. Eight weeks ago had mental strain (death of brother). Right ear said to have been normal until six weeks ago, when it began to discharge, with slight deafness. Four weeks ago, while being syringed, sudden and almost complete deafness occurred, which has remained. Occasional whistling tinnitus, but no giddiness or other symptoms.

On Examination.—Left ear: drum normal. Right ear: chronic suppurative otitis media, with extensive destruction of drum; Eustachian tubes patent on left, obstructed on right.

X-ray examination of skull reveals nothing of importance. Wassermann reaction negative.

Cochleæ.—Left: no response to any sound. Right: patient's answers always suggest great loss of hearing, but replies are very variable. Rinné is consistently negative, yet Bing is positive. Answers not altogether reliable. Lombard's test is negative. Loss of bone conduction almost equals loss of air conduction.

Voice of patient is not raised.

Stato-kinetic Labyrinth.—Cold caloric test (results complicated by presence of varying spontaneous bilateral nystagmus, probably optical). Left: no response. Right: normal responses of vertical and horizontal canals.

Patient otherwise well. No stigmata of nervous degeneration.

The case is shown because: (1) a diagnosis of hysteria is never completely satisfying; (2) it is hoped that suggestions for treatment will be made. Amateur suggestion has been used without effect.

Discussion.—Mr. A. R. TWEEDIE said he was only able to make a brief examination of the case, but noted that there was definite objective evidence of advanced middle-ear disease in the right side, and so far as he could judge the patient *did* hear a question. What were the responses to the galvanic and caloric tests?

Mr. PETERS said he thought that the right cochlea and posterior labyrinth had been damaged in the original concussion but that the posterior labyrinth had recovered. Then had come the attack of suppuration, which had considerably reduced the hearing. In a rather short examination he had found that a tuning-fork 128 gave four seconds bone-conduction with several tests.

Mr. SYDNEY SCOTT said the case gave him the opportunity of demonstrating the value of the "minimal-stimulus test" in rotation. When this patient was rotated slowly through a small angle (10 degrees) to his right, he instantly felt that he was going to his right, while if rotated through the same angle to his left he did not appreciate the movement. The patient was so consistent that he (Mr. Scott) concluded that the left vestibular nerve was defunct.

Mr. SOMERVILLE HASTINGS (in reply) said he was sorry more time could not be given to this case. The galvanic stimuli had not been worked out. The right ear was normal to the caloric tests. He brought the case because he felt that the deafness on the right side, which came on suddenly after nervous shock, was of functional origin. The man was sent to a neurologist, and every effort made to get back the hearing, but without success.

Section of Orthopædics.

President—Mr. W. Rowley Bristow, F.R.C.S.

[*March* 6, 1928.]

SPECIAL DISCUSSION ON THE TREATMENT OF ACUTE OSTEOMYELITIS.

Mr. Harry Platt (Manchester).

Preliminary Considerations.

The problems of acute hæmatogenous osteomyelitis have exercised the minds of surgeons for many generations. In the "pre-abdominal" era the suppurative diseases of bone occupied a fairly conspicuous place in the surgical emergencies of the day, and probably such conditions were treated with as great success as is attained on the average at the present time. But during the past quarter of a century acute osteomyelitis has considerably lost in importance, for two main reasons. First, with the change in surgical perspective caused by the abdominal revolution, acute osteomyelitis with other commonplace affections of the skeleton, has been relegated to the background. For the careful personal attention of the older master surgeon—who himself often dressed the wounds he created—the unregulated enthusiasm, and transitory interest, of juniors have been substituted. Indeed, in many hospitals it is the custom to assign all patients with acute osteomyelitis to the tender mercies of the surgical residents. The second reason is simpler and has no controversial implications. *Acute osteomyelitis is admittedly an uncommon disease at the present day.*

When we survey the extensive literature attached to this subject, we find its influence as a whole somewhat unsettling. Too often the surgical teaching of a particular school has been swayed in the direction of dogma, by conclusions drawn from the analysis of a comparatively small number of cases treated over a fairly long period. A typical report or contribution to a discussion is a study of some *fifty cases* in a *ten-year period*, from which it would appear that this or that operation—e.g., primary resection of the diaphysis—is the method of choice. What does such an analysis imply, if we consider it dispassionately? That the fifty patients were dealt with by almost as many different operators. In a hospital with five surgical units, this would be an average of *two* cases of acute osteomyelitis to each unit every year. We cannot of course blame any hospital, or any individual surgeon, for the scarcity of material on which reports are made, for this is a disadvantage common to all who practise surgery. But we should welcome authoritative teaching based on a large series of cases treated in a clinic with unity of control, where operations based on an agreed conception of the pathology of this affection have been carried out by the chief of that clinic and his associates. Such knowledge has lately become available as a result of the experimental and clinical studies of Clarence Starr, of Toronto, whose writings, in my judgment, form the outstanding contribution to the surgery of acute osteomyelitis during the past decade

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Incidence.

It will be instructive at this stage to deal briefly with the question of incidence. The statement that acute osteomyelitis is comparatively uncommon is well borne out by recent statistics. In the Registrar-General's report (England and Wales) for 1926, the deaths from acute osteomyelitis for the year were 417, as compared with 2,710 deaths from acute appendicitis. The mortality-rate has remained fairly constant during the six years, 1921-1926 (inclusive), the average number of deaths per annum being 435. The highest mortality occurs during the age-period 10 to 15, and the male sex predominates in the proportion of 2 to 1.

Statistics from various hospital reports also form a useful commentary on the frequency of this disease in surgical practice. At St. Thomas's, during 1925, fourteen cases were treated; in 1924, seventeen cases; in 1923 there were seven operations, and in 1922, nine operations for acute osteomyelitis. At University College Hospital, in 1927, there were twelve cases; in 1926, three cases; and in 1925, nine cases. In one of Clarence Starr's most recent articles[1] there is a reference to a series of 207 cases treated at the Toronto Children's Hospital in twelve years. The London Hospital, with its exceptional bed accommodation, and vast clinical population, is more fortunate. In 1926, thirty-five cases were dealt with, an average of seven to each surgical unit.

In the discussion on acute osteomyelitis at the Annual Meeting of the British Medical Association, in 1924, figures from St. Bartholomew's were quoted by Mr. Acton Davis.[2] In two separate four-year periods, forty-five cases were operated on by the method colloquially known as "the gutter," and seven by diaphysectomy, a total, for eight years, of fifty-two cases.

In my own unit at the Ancoats Hospital, which deals with all the acute osteomyelitis material of the hospital, I have had forty-one cases only during the past five years (1923-1927 inclusive). One might quote figures from many other sources to illustrate the comparative scarcity of such material at the present time.

In introducing the subject I shall confine my attention to certain aspects of the pathology, symptomatology, and surgical treatment. On the general ætiology, skeletal distribution, and bacteriology of the disease, there is little scope for discussion or controversy. Some of these points are illustrated in the following table:—

Forty-one Cases. Ancoats Hospital.—Bones affected: Tibia, 13; femur, 7; fibula, 4; humerus, 4; radius, 3; ilium, 4; ulna, 2; metatarsus, 2; mandible, 1.

Bacteriology: *Staphylococcus aureus*, 33; streptococcus, 4; *Staphylococcus albus*, 1; not recorded, 3.

Age-period: Under 10 years, 15; 10 to 15 years, 19; over 15 years, 7.

The term "acute osteomyelitis" implies an acute inflammatory lesion affecting all the various anatomical constituents of bone. Further, the type of acute bone infection we are considering is a metastatic phenomenon, dependent on the existence of a primary bacterial focus elsewhere in the body.

Anatomy and Blood-supply of the Long Bones.

The schematic blood-supply of the long bones, first taught by Lexer, forms a useful basis for a consideration of the avenue of entry into the bone of infective emboli, and the mode of spread of an inflammatory process (fig. 1). Lexer's earlier conclusions that the terminations of the three distinct systems of blood-vessels which supply the juxta-epiphyseal cancellous tissue (epiphyseal, metaphyseal, and nutrient

1 Starr, C. L., "Osteomyelitis," in Lewis's "Practice of Surgery," 1928.
2 *Brit. Med. Journ.*, October 4, 1924

sets) are end-arteries, has not always been confirmed by later observations. The existence of vessels running from the metaphysis through the growth disc into the epiphysis has been known since the days of John Hunter. Whether the end-artery conception be correct or not, of equal significance is the extreme tortuousity of the capillaries in the metaphysis, which leads to a slowing of the blood-current and thus favours the arrest of tiny emboli (Hobo).

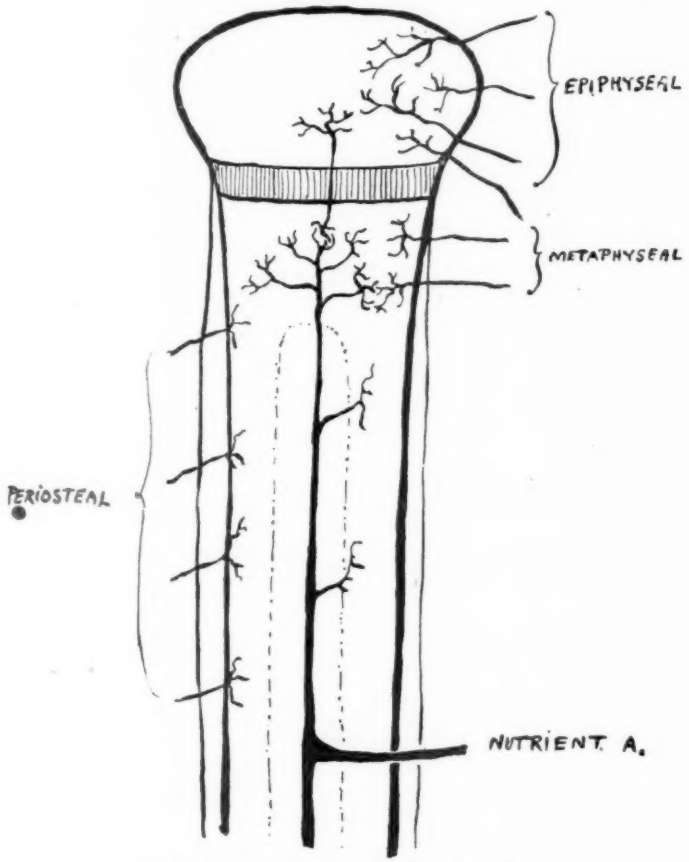

FIG. 1.—Schematic blood-supply of long bone (Lexer).

It is important for our purpose to contrast the blood-supply of the growing bone with that of the adult. In the child the dominant vascular region is the metaphysis. As the period of cessation of growth approaches, the vascular picture of the bone changes. The shaft-supply from the nutrient artery assumes greater importance, and it is also probable that free intercommunication develops between systems or merly separate.

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A good deal of experimental work has been done to determine the relative values of the various systems. The results appear to show that: (*a*) The *nutrient* artery maintains viability throughout the medullary canal and deeper half of the cortex; (*b*) the *metaphyseal* group of vessels supply the cancellous tissue of the juxta-epiphyseal region, and the deeper half of the overlying cortex; (*c*) the periosteal vessels supply the superficial layers of the diaphyseal cortex only. Repair is most active when the nutrient supply is intact, and feeblest when the bone has to depend on the periosteal supply alone (Johnson).[1] These facts have a most important bearing both on the spread of the osteomyelitic process and the rationale of the various forms of operative treatment.

Morbid Anatomy.

I.—*Primary Localization and Spread in the Growing Bone.*

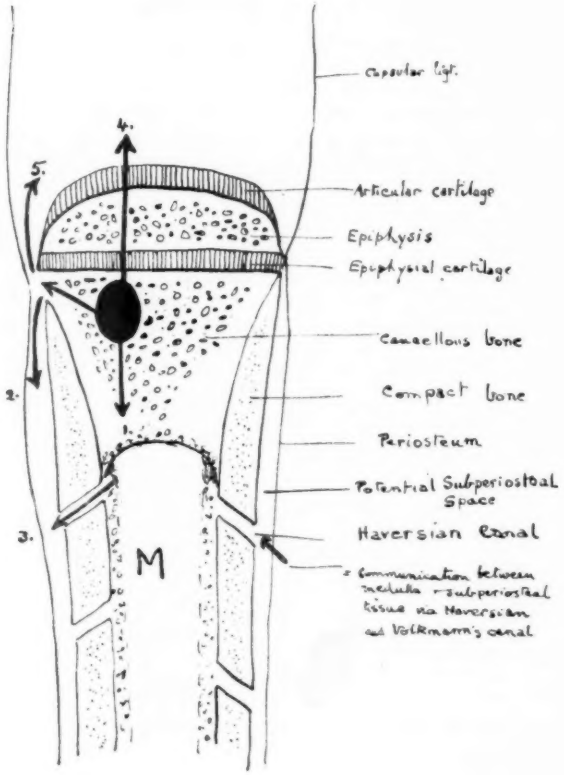
A. Metaphyseal Osteomyelitis.—It has long been established from clinical, pathological and experimental observations, that in the growing bone the acute osteomyelitic process almost invariably begins in the metaphysis. The end-artery system and sluggish blood-stream are factors which determine the lodgement of infective emboli in the cancellous tissue just beneath the epiphyseal disc. The tiny inflammatory focus which thus results may remain latent for a time, or may begin to spread with great rapidity. The possible routes of extension of the original juxta-epiphyseal focus, as usually taught to students, are familiar to all (fig. 2). Certain directions of spread are rare. These are the invasion of the neighbouring joint after the breaking down of the stout barrier formed by the attachment of the periosteum to the epiphyseal line, or from perforation of the epiphysis itself. The pathological picture which then results—i.e., an acute suppurative arthritis—lies somewhat outside the scope of our present discussion.

A most important line of advance is the tracking of the infection along the plane of the epiphysis to reach the subperiosteal space. From here there is little to prevent rapid extension along the surface of the shaft, owing to the ease with which the periosteum strips in the young bone. It has long been taught that *pari passu* with the subperiosteal spread, the infection extends downwards in the interior of the bone, invading the medullary canal for a considerable distance along the shaft. Upon this conception, now proved to be erroneous, the treatment of acute osteomyelitis by means of the "gutter" operation has been based.

The work of the Toronto School has finally settled the vexed question of the chronology of the spread of the acute osteomyelitic process in the interior of a long bone. Starr has shown that whilst the spread to the surface, and so to the subperiosteal space, is early and rapid, the cancellous tissue of the metaphysis seems to offer considerable opposition to downward spread towards the medullary canal. In fact, the juxta-epiphyseal focus remains small and localized at a time when considerable stripping of the periosteum has taken place. It is not uncommon for the whole shaft to be stripped, with the medullary canal remaining absolutely uninvaded. The medullary canal proper is rarely infected until a much later stage, and is then often invaded not directly from the metaphysis, but from the sub-periosteal space along the Haversian canals (retrograde infection). An important avenue of longitudinal spread in the interior of a pipe bone is along the cancellous tissue which lines the deep surface of the cortex.

These conclusions, supported by most convincing clinical, experimental, operative

[1] Johnson, R. W., *Journal Bone and Joint Surgery*, ix, 1927.



Schematic representation of a long bone, showing directions of possible spread of infection from the metaphysis

after Cheyne

FIG 2.

and post-mortem observations, have influenced my own attitude towards acute osteomyelitis since 1922, when Starr's first paper came before my notice.[1]

B. Diaphyseal Osteomyelitis.—In older children an atypical primary localization may occasionally be seen in certain of the long bones (e.g., radius and ulna). The infection may reach the bone by the nutrient artery and the initial focus develop towards the middle of the shaft. The subsequent spread then occurs along the cancellous lining and in the medullary canal itself towards the proximal and distal ends of the bone.

Archives of Surgery, May, 1922.

II.—*Primary Localization and Spread in the Adult Bone.*

The pathological picture of acute osteomyelitis in the adult long bone is not inherently different. It is simply modified by anatomical conditions to which we have already alluded. The primary focus may be localized: (*a*) in the middle of the shaft, when the infection has reached the bone by the nutrient vessels; or (*b*) in the cancellous tissue of the old metaphysis, as in the child. From either site extension may occur, both in the interior of the bone and along the surface. There is, however, a striking tendency for the initial focus to remain latent, and for the osteomyelitic process to spread somewhat slowly. The periosteum of the adult bone offers considerable resistance, so that when the infection reaches the surface, and pus accumulates under tension, it is not uncommon to find that a breach occurs, allowing the pus to escape into intramuscular planes. In the adult the primary focus may become shut off by a zone of sclerosis, with the ultimate formation of a chronic abscess, situated either in the centre of the shaft or at the end of the bone.

III.—*Reaction of Bone to Infection.*

The changes which occur as the osteomyelitic process spreads, represent the inevitable reaction of bone to the destructive and stimulative influence of bacterial invasion. Considerable periosteal stripping may occur without endangering the nutrition of the shaft, if the juxta-epiphyseal focus remains localized. Nor does early necrosis necessarily imply the future formation and separation of sequestra, since many areas of cortex or cancellous bone which have been completely devascularized are later reintegrated in the shaft as the infection becomes attenuated. It is obvious that such natural repair may be jeopardized by injudicious operative technique. The changes in the so-called chronic stage, in which bone formation and late necrosis ordinarily predominate, do not require detailed consideration, for the outstanding surgical problem of acute osteomyelitis is the prevention of such sequelæ.

SYMPTOMATOLOGY.

In the clinical picture of acute osteomyelitis it will be useful to dwell on those signs which are of chief importance in relation to early diagnosis and differential diagnosis.

I.—*The Prodromal Phase.*

A study of the symptomatology suggests that there are two main clinical groups: (1) Where the local and constitutional signs appear somewhat abruptly after a very *short prodromal phase* lasting two or three days. This phase may be non-existent in the clinical sense, or the patient may complain of transitory pain in the affected limb or part. With such an onset the condition develops along the accepted lines, and, according to the speed of progress and the severity of the symptoms, various clinical types are distinguished, e.g., the rare fulminating type, the ordinary severe type, and the mild or more subacute type.

(2) In the second group are found those cases of acute osteomyelitis of every degree of severity where there is a definite clinical prodromal phase lasting from seven days to three or four weeks, during which localizing signs of the greatest importance occur, but which usually pass unnoticed. In my own recent series of forty-one cases, in twenty-two there was a definite prodromal phase of this kind, and I believe the proportion would be even greater if the case histories had been more carefully taken. I have also scrutinized the records of more than 100 cases which have passed through my hands, and from the imperfect records have obtained glimpses of this phase again and again. I would therefore lay the greatest emphasis

on the prodromal stage of acute osteomyelitis, for that must obviously correspond to the stage at which the tiny juxta-epiphyseal focus is smouldering, but has not begun to spread. Of the localizing signs which characterize this phase, two are of outstanding importance: (*a*) *Fixed pain at the end of a long bone, and* (*b*) *metaphyseal tenderness.* Intermittent limp and other slight subjective or objective symptoms may also be present. A prodromal stage with a definite symptomatology is more usual in older children and adolescents, in whom an accurate history is more easily obtained than in younger children. Thus, a diagnosis should often be possible in the earliest stage of the disease—in patients between the ages of 10 and 15—the period of greatest mortality. If there is a history of a recent skin sore or other superficial sepsis, the early localizing signs take on added significance.

We must therefore correct the perspective of our clinical teaching on acute osteomyelitis, and turn away from syndrome of the disease in the florid stage to that prodromal phase in which the diagnosis should be increasingly possible, if the significance of the localizing signs were more widely appreciated.

II.—*The Early Stage.*

The local and general symptoms of the prodromal phase, at first somewhat elusive, become more insistent as the juxta-epiphyseal infection reaches the subperiosteal space. At this stage definite *bony swelling* is added to the metaphyseal tenderness, and occasionally there may be a slight effusion in the neighbouring joint. Accompanying these local signs are the symptoms of a general infection, mild, or severe. When such a clinical picture is established, the diagnosis should rarely be in doubt, but, owing to the comparative infrequency of the disease, the syndrome is commonly regarded by the practitioner as indicative of some other acute infection.

In the average acute osteomyelitis arising late for surgical treatment, a diagnosis of acute rheumatism has usually been made. It is an easy matter to enumerate the points of difference between acute osteomyelitis and acute rheumatism. At first sight there is very little resemblance between the clinical picture of the two infections. An acute synovitis with *joint* pain, joint tenderness and muscular spasm, is in striking contrast with the painful, tender, and thickened *metaphysis*, even though the latter is in close proximity to a joint. Whilst we must continue to emphasize these distinctions, it is clear that the student and practitioner would be considerably aided if the incidence of acute monarticular rheumatism were defined. Is there such a clinical entity between the ages of 10 and 15? and if so, is it common or rare? If acute monarticular rheumatism be rare, or non-existent, then acute osteomyelitis must be given pride of place in the clinical consciousness of the practitioner. When such a changed attitude of mind becomes general, acute osteomyelitis will be suspected both in the prodromal stage and in the earliest phase of subperiosteal spread. It will then be possible in all doubtful cases to perform an exploratory operation. A small incision down to the metaphysis at the point of tenderness, and puncture of the bone by means of a series of drill holes, will save many limbs and many lives. If the diagnosis be unconfirmed the patient will suffer no harm.

III.—*The Early Diagnosis of Acute Osteomyelitis in Deeper Bones* (*Pelvis, etc.*).

So far, the clinical picture of acute osteomyelitis has been considered in relation to the long bones. Special difficulties are encountered in the diagnosis of acute infections of the flat bones—more particularly of the pelvis. The mortality in acute osteomyelitis of the pelvis is very high. From the Moscow Children's Hospital, Krasnobaew records twenty cases out of 428 cases of acute osteomyelitis in twenty-one

years. Of the twenty, eight died (40 per cent.).[1] In pelvic osteomyelitis the ilium is the usual site, but we have little knowledge concerning the primary localization of the infection. When it begins near one of the epiphyseal lines there is usually an early extra-osseous spread; rupture of the periosteum occurs and a large abscess tracks amongst the surrounding muscles. In other cases the inflammatory process extends widely in the diploë, and does not reach the surface.

Localized pain and deep tenderness may be absent or inconspicuous in the early stages of acute osteomyelitis of the ilium. In the presence of an acute toxæmia suggesting osteomyelitis, but without definite localizing signs, the ilium should always be suspected. There are three suggestive clinical signs which may assist in diagnosis: (*a*) Referred pain radiating down the thigh on the same side; (*b*) pelvic tenderness on rectal examination; (*c*) a sense of deep resistance between the trochanter and iliac crest. In the two fatal cases out of four examples of acute osteomyelitis of the ilium in my last series, referred pain was a well-marked symptom in the prodromal and early stages, but its significance was appreciated only in the retrospect. It is a symptom which can hardly be fortuitous, and one which I shall always bear in mind. I would urge the necessity of exploratory puncture of the ilium, without delay, in every case of suspected pelvic osteomyelitis.

IV.—*Differential Diagnosis of Subacute Osteomyelitis.*

In the adolescent or adult, the course of hæmatogenous osteomyelitis is not infrequently subacute, with a resulting clinical and radiographic picture which may simulate other chronic bone affections, notably sarcoma. The differential diagnosis of such conditions opens up a wide field for discussion which lies outside our immediate objective.

Treatment.

A consideration of the mode of origin and spread of the osteomyelitic process in a long bone inevitably leads us to enunciate certain principles which should govern both the local and general treatment of the early lesion and its ravages. I would state these as follows:—

(1) It should be the aim of the surgeon to establish early and effective drainage, and to achieve early sterilization of the wound.

(2) The operation must be so designed that: (*a*) further devascularization of both infected bone and uninfected bone is avoided; and (*b*) infection is not directly introduced into uninfected areas.

(3) The systemic infection must be combated by appropriate general and specific treatment. In severe toxæmia or septicæmia, intravenous antiseptic medication and immuno-transfusion should be practised. Into the minutiæ of such therapy I do not propose to enter.

Operative Treatment.

In the surgical treatment of acute osteomyelitis three main types of drainage operation are commonly practised at the present time.

I. *Subperiosteal and Metaphyseal Drainage.*—It is obvious that in the earlier stages drainage of the infected area alone, implies: (*a*) drainage of the cancellous tissue of the metaphysis, combined with (*b*) drainage of the subperiosteal space in the area of spread. Although a subperiosteal collection of pus develops only when the infection has escaped through a definite hiatus in the bone, drainage of the subperiosteal space alone is insufficient, for the hiatus is always minute and often hidden on the deeper aspect of the bone. In patients who are desperately ill with

[1] *La Chirurg. degli Organi di Movim.*, 1926, July.

a large extraperiosteal and subperiosteal collection of pus, and especially in osteomyelitis of the flat bones, it is occasionally necessary to be content with extra-osseous drainage alone as a temporary measure.

In the average case of acute osteomyelitis, however, it is sound practice to open freely into the metaphysis, either by multiple drill-holes, after the original technique of Starr, or by removal of a small trephine disc. All pus and infective débris should be sponged out lightly, but curettage of the interior should be avoided.

At this stage there cannot be the slightest justification for the common practice of extending the opening into the bone along the shaft, and exposing the medullary canal for a distance equivalent to the limits of the subperiosteal stripping. (*The gutter operation.*) We should unite to condemn this procedure and to explain its futility and dangers when the osteomyelitis is confined to the metaphysis.

Where the method of conservative drainage of the metaphysis has been consistently practised in acute osteomyelitis, the results have been most convincing. When carried out at the stage at which the periosteal stripping is minimal, or where the infection has not reached the surface, healing should be obtained without subsequent sequestration. In 207 operations at the Toronto Children's Hospital, twenty-six healed in this manner. This technique has been followed as a routine in my own clinic for the past five years. In the forty-one cases which I have already quoted, subperiosteal and metaphyseal drainage was performed in twenty-two. Fourteen of these healed without sequestration ; in the remainder, sequestra of various shapes and sizes were extruded spontaneously or were removed at a secondary operation.

II. *Diaphyseal Drainage* (*The Gutter*).—The gutter operation has its legitimate indications when the medullary canal with its lining of cancellous bone is infected widely. Such conditions are seen in osteomyelitis originating in the centre of the shaft, or in neglected cases of primary metaphyseal infection. Where the osteomyelitic process has travelled extensively in the cancellous tissue of the shaft of a pipe bone, it is difficult to establish drainage without imperilling the viability of the shaft as a whole. In such circumstances the gutter operation must tend to add to the amount of necrosis already determined by vascular obliteration and the chemical action of continued suppuration. In bones with a considerable subcutaneous area, e.g., the tibia, the gutter has also the great disadvantage of leaving an avascular adherent scar. In my last series there were four operations of the gutter type, with one death. In one of the remaining three cases healing occurred without sequestrum formation, and in the two others extensive necrosis resulted.

III. *Subperiosteal Resection of the Diaphysis.*—Here we are on more debatable ground. If this operation is to be justified as a method of primary drainage, it must fulfil certain requirements. The arguments in its favour are: (*a*) That it allows a most effective drainage of the subperiosteal space ; (*b*) that infected bone which would undergo massive sequestration at a later stage is removed at the outset, and thereby prolonged suppuration and secondary operations are avoided. In theory both arguments are sound. The obvious objection to the operation is that it is likely to be too radical as a method of simple drainage, and that complete or partial failure of regeneration of the shaft, with all its sequelæ, may be seen under certain conditions. On the rôle of diaphysectomy my mind is, in the words of the late President Woodrow Wilson, "open and to let," although in the past fifteen years I have practised it only in some half-dozen cases of acute osteomyelitis. In my last series (forty-one cases) there was one diaphysectomy. For the earlier stages of acute osteomyelitis, where the results of conservative metaphyseal drainage are beyond criticism, diaphysectomy can have no justification ; in the neglected case, where the whole shaft is riddled with infection, the operation is, however, in theory more

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rational than the gutter. But whenever performed in *acute* osteomyelitis, there must always be a definite risk of non-regeneration, owing to destruction of the periosteal tube in part or as a whole. In the less acute types failure of regeneration need not be feared, a fact which suggests that the operation is more suitable in the treatment of *subacute* osteomyelitis.

There is one final consideration in assessing the value of any operative procedure in acute osteomyelitis. It is customary to compare the respective mortalities of the various operations—a most illogical form of argument. In many cases of acute osteomyelitis, when first seen by the surgeon, there is a profound septicæmia and the fate of the patient is not determined by operative treatment.

Conclusions.

(1) A close study of the fleeting symptomatology of the prodromal phase of acute osteomyelitis will lead to earlier diagnosis.

(*a*) Fixed pain at the end of a long bone, and (*b*) metaphyseal tenderness are the most significant early signs. In all doubtful cases exploratory puncture of the bone should be made.

(2) With early diagnosis the operation of conservative drainage of the metaphysis becomes the correct routine. The gutter operation and diaphysectomy should be needed only in advanced and neglected cases.

Mr. Alexander Mitchell.

Acute osteomyelitis is one of the most serious clinical conditions with which we have to deal, and, although in most cases the local treatment is of vital importance, in a certain proportion the accompanying septicæmia is so virulent that the outlook is hopeless from the start.

In every case a blood culture should be taken at the first operation. It is obvious that this is desirable, but it is often omitted.

Generally speaking, the most important factor in a favourable prognosis, both as to life and to future function, is early treatment, but this is not always possible.

As to actual operative treatment there is considerable diversity of practice, from simple incision through the periosteum, followed by fomentations, to resection of the complete diaphysis. Of simple incision I had some experience as house surgeon in a children's hospital and the results were disastrous. A possible exception to this may be in the mild type of pneumococcal osteomyelitis seen in young children.

In an early case of septic osteomyelitis of the end of a long bone, taking out a piece of the cortex to provide for drainage, gives in most cases a good immediate result, but in a certain proportion secondary operations are needed, prolonged suppuration ensues, and the end results are not uniformly perfect. Judging from the results of my own and other cases, I do not think that subperiosteal resection of all the length of bone affected has received the recognition it deserves, but I realize that there is a sharp cleavage of opinion on this point. In cases with an extensive infection of the tibia, humerus, or femur, I have not found drainage of the end or "guttering" of the length of the bone satisfactory; the tibia and humerus readily become infected in all their length, the femur in the lower half or two-thirds. In such cases it has been for many years my practice to resect the whole length of the infected shaft, and I believe that my results justify this procedure. Rarely resection may result in the production of an irregular and unsightly bone, and in one case I have seen complete failure of regeneration; but such a result occurs only in virulent ases and may be regarded as an alternative to amputation. In over 90 per

cent. of cases, with careful after-treatment, securing extension and early active muscle exercise, a good result will be ensured. An argument against resection is that it is attended by a high mortality. In my last seventy cases primary resection was done thirteen times, with one death, and the patient in that case had active septicæmia. The total mortality from these seventy cases was ten—a very high one—I think explained by the fact that of these ten, eight had an active blood infection. Patients treated by more conservative methods when the disease has been extensive have to undergo additional operations for the removal of long sequestra.

Recently I have had two such cases, with long sequestra in the femur. In the first case the patient suffered so much from hæmorrhage, in spite of great care in packing the wound during operation, that a blood transfusion was necessary, and she narrowly escaped with her life. The second patient died an hour after operation from a pulmonary embolism. Secondary operations may therefore be dangerous, and the longer the course of the suppuration the worse the functional result is likely to be. I do not wish to stress subperiosteal resection too much, but I think it has a place, and that with experience one can recognize cases suitable for treatment by this method.

In several cases I have tried blood transfusion, but only once it appeared to be beneficial. That was at a late stage in a hæmolytic streptococcal case, and the good result was due mainly to the resulting improvement in the anæmia. In acute blood infection such as one meets with in osteomyelitis or in pneumococcal peritonitis I have never seen any permanent good result from blood transfusion.

I rarely see a case of severe osteomyelitis in my private work, but, although the cases are nearly all hospital ones, many of the victims are well-nourished, healthy-looking children, and one would expect children of the hospital class, when healthy, to be more resistant to sepsis than those leading a more sheltered life.

It is our duty to impress on our students the gravity of this disease, the importance of early diagnosis, and the value of a minute examination of the ends of the long bones in every child who has a persistent rise of temperature which cannot be readily accounted for.

Mr. Eric Lloyd

said he had collected and analysed a series of twenty-nine cases in the five years 1923-27 at the Hospital for Sick Children, Great Ormond Street.

He had been rigorous in excluding subacute cases and by so doing he felt that perhaps he had scarcely done justice to his hospital. The series, though small, derived some additional interest from the fact that the majority had been under his own observation, though by no means all had been in his care. Over two-thirds of them had been seen by him and he had operated upon rather less than half the total number himself. Thus there was an element of uniformity which large series sometimes lacked. Table I showed the greater frequency with which the lower limb was affected. Table II illustrated the high mortality associated with *Staphylococcus aureus* compared with that associated with other organisms. In Table III the eleven fatal cases were analysed and it was seen that the heart or pericardium had been involved in four out of seven of these.

Treatment had usually consisted in removal of about two-thirds of the circumference of the shaft of the long bones over such length as appeared diseased. This was something more than the "guttering" operation and only left enough bone to act as an internal splint. This entailed a risk of fracture during or after operation

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TABLE I.

	Recovery	Died	Total
Ilium	1	1	2
Pubis ischium	2	0	2
Femur	1	5	6
Tibia	3	1	4
Fibula	1	2	3
Scapula	2	0	2
Rib	1	0	1
Humerus	3	2	5
Radius	1	0	1
Ulna	1	0	1
Spine	1	0	1
Skull	1	0	1
	18	11	29

TABLE II.

	Recovery	Died	Total
Staphylococcus aureus	9	9	19
,, *albus*	1	0	
Streptococcus	7	0	7
Pneumococcus	1	0	1
No culture	0	2	2
	18 (62 %)	11 (38 %)	29

TABLE III.—FATAL CASES.

	Age	Bone	Duration before admission	Wound culture	Blood culture	Duration of illness	Post-mortem examination
J. S. ♂	8	Ilium	5 days	*Staphylococcus aureus*	*Streptococcus*	59 days	Both hips dislocated. Large abscess communicating with right hip-joint
M. B. ♀	7	Femur (upper third)	4 ,,	,,	*S. aureus*	48 ,,	No P.-M. Pericarditis (clinically)
M. L. ♀	3	,,	4 ,,	,,	*S. albus*	8 ,,	Local P.-M. only
J. G. ♀	4	Femur (shaft)	1½ ,,	,,	—	9 ,,	No P.-M.
I. M. ♀	3	,,	2 ,,	,,	—	3 ,,	Heart normal
P. S. ♂	2	,,	2 ,,	,,	—	3 ,,	No P.-M.
H. D. ♀	½	Tibia	3 ,,	,,	—	13 ,,	Pyopericardium
R. W. ♂	4	Fibula	3 ,,	,,	—	8 ,,	No P.-M. Pericarditis (clinically)
A. E. ♂	4	,,	2 ,,	,,	*S. aureus*	6 ,,	Ulcerative endocarditis with perforation of mitral valve
C. M. ♂	½	Humerus	7 ,,	Sterile	—	35 ,,	Died from streptococcal meningitis
M. D. ♀	¾	,,	½ ,,	No culture	—	1 ,,	No P.-M.

and such fracture had occurred. It was his practice to open the bone and never to be content with incision of the periosteum. The usual after-treatment had been irrigation with 2 per cent. chloramine through Carrel-Dakin tubes and the wounds

had been left open. Amputation had not been performed in any case. Possibly treatment nowadays was too conservative.

His experience with transfusion and intravenous medication had been disappointing, three patients had had blood transfusion (two of these were immuno-transfusions) and all had died. One recovery had followed intravenous saline. Blood transfusion was not of any value in these cases.

Intravenous perchloride of mercury had been used on one or more occasions in four patients, with one recovery (a streptococcal septicæmia.) Intravenous injection of drugs was often worth trying. The staphylococcal septicæmias were lethal and he had never seen a patient recover in such cases, though he had seen patients with streptococcal and pneumococcal septicæmias make surprising recoveries.

Diaphysectomy had only been performed in one patient in this series, and though the result was gratifying he felt that as the infecting organism was a streptococcus this materially helped to ultimate success.

[Mr. Lloyd showed a series of lantern slides of the skiagrams taken in this case. The upper four-fifths of the diaphysis of the humerus had been removed three years previously and excellent regeneration had followed. For comparison with this patient, Mr. Lloyd showed skiagrams from a similar case of Mr. Addison's. In that case the patient had been aged 4½ years; his own patient (originally under Mr. Waugh's care) was aged eight months. In each case there was streptococcal infection of the humerus; diaphysectomy had been performed in one and the modified gutter operation in the other.]

He wondered whether the high mortality amongst patients in whom the infection began in the upper third of the femur was a general experience.

Mr. W. H. Ogilvie.

Omitting infections of bone due to injury and to spread of disease from adjacent parts, fifty-one cases of acute osteomyelitis were admitted to the surgical wards at Guy's Hospital during the five years from 1922 to 1926 inclusive. The number is small, but we are finding, in common with most London hospitals, that osteomyelitis is a vanishing disease. Now that dirt, destitution, and drunkenness are disappearing from South London, it is going the way of chlorosis and other conditions based upon diminished resistance.

Table I.—Acute Osteomyelitis.

Admissions to Guy's Hospital 1922-1926.

Total number		51
Sex incidence.	Males	39 or 80 per cent.
	Females	9 or 20 per cent.
	Not stated	3
Age incidence.		
Average age in the series		12·9 years
Average age in children		10·25 years

(Four adult cases over 21 omitted.)
Greatest incidence at age of 13.
68 per cent. of all cases between the ages of 8 and 15.

A preponderance of males is found in all published statistics. Choyce gives the proportion as three to one, Fraser as six to one. The age incidence in the Guy's series tallies with that given by the majority of writers, though in the figures of Trendel, quoted by Whitman, the period is said to be from the thirteenth to the seventeenth year.

TABLE II.—INCIDENCE OF PRIMARY FOCUS WITH REGARD TO BONES.

Tibia.	Upper end	8
	Lower end	8
	Whole shaft (probably starting upper end)	7
	Both tibiæ	1
	Total	24 or 47 per cent.
Femur.	Upper end	0
	Lower end	12
	Total	12 or 23 per cent.
Humerus.	Upper end	3
	Lower end	1
	Total	4 or 8 per cent.
Fibula.	Upper end	3
	Lower end	1
	Total	4 or 8 per cent.
Ilium		3 or 6 per cent.
Os calcis		2 or 4 per cent.
Pubis		1 or 2 per cent.
Metatarsals		1 or 2 per cent.

Here the order of frequency—upper end of tibia, lower end of femur, lower end of tibia, upper end of humerus—is the same as that given by Fraser. The comparatively large number of infections of the fibula is one of those chance variations which must appear in any small series.

An examination of the results in this series brings out the well-known high mortality of the disease, and the crippling which it leaves in those who survive.

TABLE III.

Results.

Died	11 or 21 per cent.
Discharged with healing wounds	26 or 51 per cent.
Discharged with wounds healed (including one case of partial suture and one case of *Bacillus typhosus* infection)	8 or 16 per cent.
Discharged with amputations	4 or 8 per cent.
Transferred to fever hospital	1 or 2 per cent.
Transferred to infirmary	1 or 2 per cent.

TABLE IV.—ANALYSIS OF MORTALITY.

(1) *According to bone involved.*

Ilium	33 per cent.
Tibia	25 per cent.
Humerus	25 per cent.
Fibula	25 per cent.
Femur	16·7 per cent.

(2) *According to type of operation.*

Radical	11 with 2 deaths—18 per cent.
Limited	27 with 6 deaths—22 per cent.
Drill holes	4 with no deaths—0 per cent.

(Three required further operation)

Incisions down to bone	6 with 1 death—17 per cent.
Incisions: pus not found	2 both died—100 per cent.

(3) *According to time between onset and first operation.*

Opened two days after onset	4 deaths
Opened three days after onset	2 deaths
Opened four days after onset	2 deaths
Opened seven days after onset	3 deaths

(4) *According to previous injury or infection.*

18 cases with previous history of injury	mortality 33 per cent.
33 cases with no history of injury	mortality 15 per cent.
7 cases with history of recent infection	mortality 14 per cent.
44 cases with no history of infection	mortality 23 per cent.

These figures emphasize the main factors which influence mortality. Disease in the ileum is fatal in a high proportion; in the lower end of the femur it is relatively benign. The good prognosis of osteomyelitis in this last situation is due to the fact that over the popliteal surface of the femur the compact bone is not only thinner than any part of that enclosing the shaft of the tibia or humerus, but is perforated by large vascular foramina; since the infection starting in the metaphysis can thus reach the surface more readily, tension is relieved and diagnosis possible at an earlier stage of the disease.

As it has not been possible to classify accurately the types of operations which were performed in these cases, I have called "radical" those operations in which the bone has been widely removed, and "limited" those in which drainage only has been attempted. When it is remembered that the radical operations have been done in the cases of wide infection, the milder ones being treated by a more conservative type of drainage, these figures seem to suggest that wide removal of bone definitely reduces the mortality. Drilling of holes has been practised only on a very few mild cases, and has not been particularly successful. The last two operations, where incisions down to bone failed to discover pus, were desperate measures in cases of a fulminating type.

The mortality depends far more upon the severity of the infection than upon any other single cause. This is seen from the next figures, Table IV (3) which give the time between the onset of symptoms and the first operations, and which show that those cases where the clinical course has been rapid, indicating a virulent organism or a poor resistance on the part of the patient, have accounted for the greater number of deaths. The same conclusion is reached from a study of Table V, in which the average duration of symptoms before operation is seen to be 4·5 days in the cases which ended fatally, as against an average of 7·4 days for the whole series. The analysis in Table IV (4) appears to indicate that those cases in which infection can be traced to a definite source, such as boils or tonsillitis, are of a less dangerous type than those in which it is due to a general enfeeblement, allowing chance organisms, which have gained access to the tissues, to establish a foothold.

TABLE V.—TIME BETWEEN ONSET OF SYMPTOMS AND OPERATION.

Average	7·4 days
Four adults	18·25 ,,
Leaving children	6·3 ,,
Ten fatal cases, average	4·5 ,,

TABLE VI.—TIME IN HOSPITAL.

Average	Two months twelve days
Omitting deaths and two transfers ...	Three months

Tables V and VI emphasize two well-known facts concerning osteomyelitis—the delayed and usually benign course taken by the disease in adults, and the burden which such cases impose upon the accommodation of any hospital.

TABLE VII.—ORGANISM.

Staphylococcus aureus	42 or 92 per cent.
S. aureus and *B. proteus*	1
S. albus	1
B. typhosus	1
Not cultivated	6
	51

In Fraser's series at Edinburgh, the staphylococcus was found in only 78 per cent., and the pneumococcus in 14 per cent. In all London hospitals pneumococcal osteomyelitis is very rare, and staphylococci are found in about 90 per cent. of the cases.

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The last table shows the incidence of the foci of secondary infection, and the secondary operations which were performed.

TABLE VIII.

Site of Secondary Infection.

Joints	11
Other bones	9
Pericardium	3
Heart	6
Lungs	3
Pleura	2
Kidney	2
Meninges	1
Soft parts	3
Local tracks	2

Late Operations.

Further operations on same bone	16
Operations on other bones	10
Amputations (two of which fatal)	5
Arthrotomy	4
Aspiration of joints	3
Opening abscesses in soft parts	3
Reduction of dislocation	1
For secondary hæmorrhage	1
Drainage of pericardium	1

My personal practice in the treatment of acute osteomyelitis is based upon those simple principles which have been borne in upon me by experience.

The first, on which I would lay particular stress, is that it is impossible at the time of operation to estimate the extent of present or future necrosis in an infected bone. That which is found bare, white, and apparently dead, may survive, at any rate in part, and form the basis of repair. Sequestration does not necessarily follow the most widespread infection of the shaft. Figures 1 and 2 are radiograms of a severe case of osteomyelitis in a boy who was under my care at Guy's Hospital in 1921, and who is now at St. Vincent's Orthopædic Hospital. When he was at Guy's, this boy had infection in five of his long bones, and pus in both knee-joints. Altogether he has had foci in about thirty different places, and among other complications an infection involving the right lateral mass of the atlas, giving rise to a large vertebral aneurysm in the occipital region. Although the destruction has been widespread, at no time have any sequestra been formed.

It is equally impossible to be certain from the appearances at operation that a bone is healthy and will survive, or to decide upon these grounds where resection should stop. However wide or however limited the operation, bone death always seems to occur later at the edge of the cut area. The whole bone is infected, and when to this infection is added the trauma of mechanical injury, death results.

The second principle is that in any operation for osteomyelitis we should take into consideration not only the immediate problem of infection, but that of the repair that will follow. All bone which does not die is a potential source of bone cells, and will help to bridge the gap left by bone that has been lost. If wide resection cannot ensure getting beyond the limits of future necrosis, it is not only useless but harmful. Resection carried to its logical conclusion means diaphysectomy, a procedure which any thinking surgeon must condemn. There are those who support it because it offers a prospect of rapid healing. A series of cases shown at a surgical meeting last year had been successful from this point of view, but had an average shortening of about an inch. This is a small criticism of the method. But non-replacement of bone, which may undeniably follow the operation, is an irreparable tragedy.

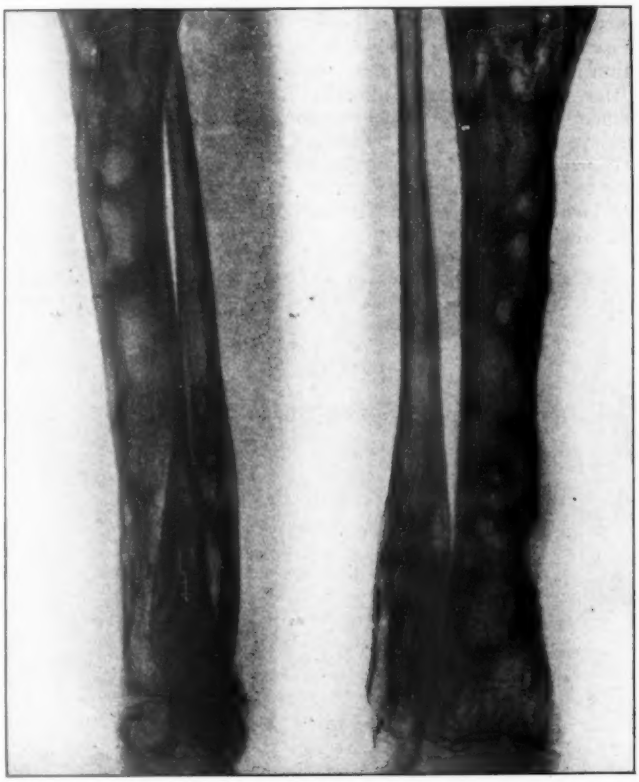

FIG. 1.

The third principle, is that drainage, and drainage only, is the immediate necessity. The infection in osteomyelitis is of a comparatively mild type, only giving rise to severe symptoms because it occurs in a cavity with rigid walls, only dangerous because the blood-vessels in that cavity are sinusoids with thin walls, through which pus can reach the general circulation. The demands of free drainage require that the medullary cavity be laid open for the length that is infected, but that is all that is immediately necessary.

The most harmful feature of the operation for osteomyelitis as commonly performed is the extensive wound kept open with packing, with the painful dressings and protracted healing which such a wound implies at the time, and the wide scar adherent to underlying bone and always ready to break down, which follows its use.

The points which I consider important in the operation are these: The incision is made in the form of a wide flap, that for the tibia, for instance, lying half an inch behind its inner border. In this way the resulting scar does not lie immediately over the bone. The compact bone only is removed for one third of the circumference,

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and in length as far as pus is found in the medulla, but no further. Wider resection is unnecessary and may leave a weak bone. Less wide may delay healing, for a deep and narrow cavity or a series of drill holes cannot fill up for mechanical reasons, even when infection is overcome, while a flattened trough can always close by approximation of the soft parts. The infected medulla is not curetted or in any way disturbed, since scraping may dislodge septic emboli into the blood-stream. The wound is not packed, but Carrel tubes are laid along the trough in the bone, and the skin incision closed by interrupted sutures one inch apart. Intermittent irrigation with chloramine T is commenced as soon as the patient gets back to bed, and is continued at two-hourly intervals till the pulse and temperature return to normal. This method, which has all the advantages of an open incision as regards freedom of

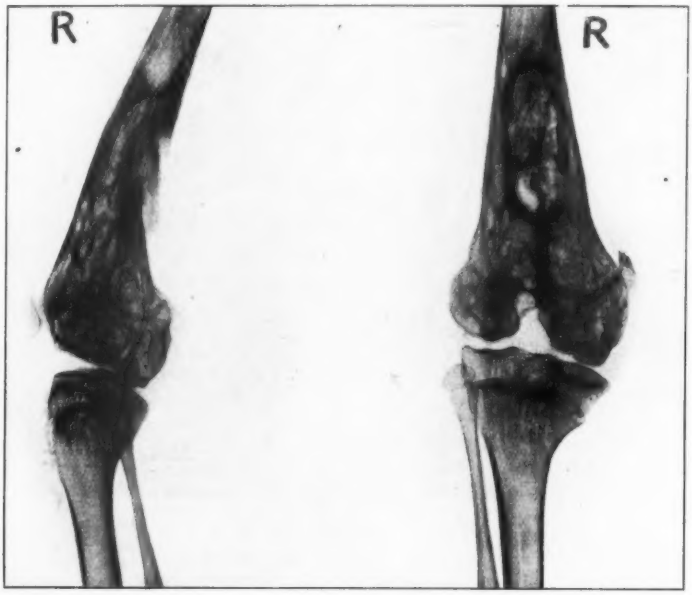

FIG. 2.

drainage, with those of suture as regards the subsequent scar, leads to early healing in a high proportion, and gives results which are more permanent than those of limited drainage.

I am sceptical about the value of radical operation in late osteomyelitis. Where there is a single cavity of simple shape in the middle of the shaft of a long bone, wide removal of one wall, allowing the soft tissues to come into contact with the base of the cavity, is very successful. The majority of radical operations succeed long enough to allow the case to be shown at a surgical meeting, but relapse inevitably. In these the original shaft is replaced by an irregular formation of new bone in which organisms are present, latent, but viable. There is no single cavity, but a network of tracks united by infected granulation tissue. Such cases run a regular course, only giving trouble when pus is formed under tension. Then the patient first

experiences aches in the bone, accompanied by a general feeling of ill health. The pain becomes steady and throbbing, there is an irregular pyrexia, and finally an abscess forms and finds its way to the surface at the old scar. Once this has happened, all symptoms disappear, the wound heals rapidly, and the patient remains well for anything from six months to ten years, when the same series of events is again enacted. In such cases I feel strongly that no operation is justified, except for the removal of definite sequestra, and for the drainage of abscesses.

Where pyrexial attacks are frequent, I have often had recourse to the method of an older generation—the institution of permanent drainage. After an abscess has opened and the discharge dwindled to nothing, I put in a length of 10-gauge silver wire, leading from the skin to the bone, and turned at the surface in a small loop. This is kept in position by strapping, and removed daily for boiling. Along such a track a serous discharge only comes out, enough to produce in twenty-four hours a stain the size of a sixpence on a piece of lint. But tension in the deeper parts cannot occur, and the pyrexial attacks are definitely and permanently abolished.

I should like to express my thanks to Mr. K. Parsons, F.R.C.S., Surgical Registrar, Guy's Hospital, for collecting the surgical reports upon which this analysis is based.

Mr. Naughton Dunn

said that two months ago he had seen a child in the out-patient department with curved limbs and a contracted spine. She had a high temperature, and a few days after admission into hospital a swelling developed in the right side of her abdomen. The general surgeon diagnosed appendicitis, but at the operation found a psoas abscess, due to the *Staphylococcus aureus*.

He asked whether there was any special method of approach in dealing with osteomyelitis of the spine.

Mr. Alan Perry

said that at London Hospital most of the cases of acute osteomyelitis came into one ward, which had been under his care for five years; the number averaged between thirty and thirty-five a year. For five years he had been looking after that ward.

Mr. Platt seemed to have had an unusual experience with regard to early cases. At London Hospital, when the tibia and humerus were involved there was widespread disease in both.

A blood-culture in 75 per cent. of cases was positive. His own view was that osteomyelitis was, largely, a pyæmia from the beginning, there being a general blood infection, rather than as a localized disease of bone.

In his hands complete diaphysectomy had been a complete failure, as in most of the cases in which it had been performed regeneration of bone had not taken place. He remembered a case in which diaphysectomy was performed on both forearms; on the radius on one side, and on the ulna on the other. The patient had recovered, but neither of these bones had regenerated. The view held at his hospital he believed, was that diaphysectomy was not warranted, a more limited operation being more successful, because bone which was left acted as a stimulus to fresh formation.

Mr. Harry Platt (in reply)

said that acute osteomyelitis of the spine offered the greatest difficulty in diagnosis. He had seen only two cases during the last fifteen years. When the diagnosis was established there was what appeared to be a large abscess arising from the transverse process or on one side of the vertebral body.

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There was not much essential difference between the views he had put forward and those of Mr. Mitchell. He felt certain that in many, if not indeed most cases of acute osteomyelitis of the long bones in children, the primary focus in the metaphysis did not extend down the shaft quickly, because it came out readily to the surface. That represented the early stage of the disease in most of the cases. Hence drainage procedures should include drainage of the infected area only, the interior of the metaphysis and the subperiosteal space as far as it had been stripped up.

With regard to the method of drainage by means of multiple drill-holes, as used in the first instance by Clarence Starr, that had now been modified by Mr. Starr—for in an article recently published he spoke of cutting a small window. He (the speaker) usually made a small trephine, but had had very good results from drilling when the diagnosis of the disease had been made in the early phase, before subperiosteal stripping had taken place, and when pus appeared at the first drill-hole. In those cases healing occurred in a few weeks, and there was no sequestration. It was at this stage that diagnosis was most important. There should be correlation of those changes with the clinical signs by a much more careful inquiry concerning the prodromal phase. Note must be taken not merely of the symptoms voluntarily indicated by the patient, but also of those which only emerged on close cross-examination and inquiry. It would often transpire that there was a fixed pain in the end of the bone, intermittent and fleeting, and varying in intensity. This fact should be impressed upon both students and practitioners. Spread occurred late along the cancellous tissue lining the pipe bone and in the medullary cavity itself. When operation was conducted at that stage, naturally drainage must be more extensive. Subperiosteal resection—diaphysectomy—when the bone was widely infected, was a first-class drainage operation, but it should be unnecessary.

The "gutter" operation also had its place, particularly in the subacute stage of the disease, when the infection had begun in the middle of the shaft and extended up and down. For the radius and ulna, of the adult especially, the "gutter" was a good operation.

Each procedure had its place; all represented an attempt to establish adequate drainage and to make as small as possible the later sequelæ which were still too familiar.

He possessed little accurate information as to the effects of blood transfusion, but in his experience patients who had been desperately ill from the disease had rallied for a time after blood transfusion had been carried out.

A blood-culture should be carried out as a routine procedure.

Section of the History of Medicine.

Président—Mr. WALTER G. SPENCER, O.B.E., M.S.

[*May* 18, 1927.]

A Pharmaceutical Fragment of the Tenth Century in Hebrew, by Shabbethai Donnolo.

By J. LEVEEN.

(*Department of Oriental Printed Books and Manuscripts, British Museum.*)

DURING the Dark Ages most of the medical works in current use in Europe were copied or miscopied from more ancient records. The earliest compositions, such as the *Commentarium Medicinale* of Benedictus Crispus of Milan (died 735), or the *Hortulus* of Walafrid Strabus (807-849) were but rearranged ancient material. More original are the Anglo-Saxon leech books certain of which may have been composed in the tenth century. Except for these the only original medical composition of the Dark Ages that has survived is the fragment of a medical work in Hebrew by Donnolo. It is interesting to note that this work is about contemporary with the earliest of the Anglo-Saxon leech books.

The name Donnolo is the Italian form of Domnulus—itself a diminutive of dominus. In the preface to his Hebrew commentary called *Hakhmōnī* on the celebrated Kabbalistic tract *Sēpher Yĕẓīrāh* ('Book of Creation') he gives details of his life and activities. From these we learn or infer that Shabbethai, called Donnolo, the son of Abraham, was born in Uris or Ures in 913. This name has been identified with Oria, a town in the South of Italy in the province of Lecce, some twenty-two miles W.S.W. of Brindisi. In those days the Byzantine Empire held the peninsula of Apulia, though the protection it extended to its inhabitants was more nominal than real. The Saracens were continually raiding the coast, and in the year 925, when Donnolo was twelve years of age, they descended on his birthplace, slew ten of the leading Jews of the town, and carried off the youth with his parents and relatives into slavery. Fortunately, Donnolo was ransomed at Otranto, but the others were transported to Palermo and North Africa to be sold as slaves.

Such conditions were scarcely favourable to learning, yet at the age of twelve we find Donnolo already deeply immersed in study. After narrating the harrowing events in which he was involved and his return to Italy, Donnolo proceeds thus:—

> I alone was left in the land of the Romans and I perceived that wisdom was more profitable than foolishness. Therefore did I labour hard to understand the science of medicine and the science of the stars and the planets. And I made copies for myself from the ancient books of Israel. But I found no Jewish scholar who understood these sciences. Certain Jewish scholars declared to me that the Jewish writings on the planets were of no value, because forsooth they did not understand them. They also assured me that the books written by the non-Jews (goyim) were quite different from the Jewish treatises. So I turned hither and thither and travelled in order to discover and study the wisdom of the Greeks, and the wisdom of the Ishmaelites (i.e., the Arabs), and the wisdom of the children of Babylon and India, and I did not rest until I had made copies of the books of Greece and Macedon in their script and in their tongue and according to their own explanations, and I also compiled from the books of the wise men of Babylon and India. And when I had diligently perused them I found them to agree in every way with the Jewish books on the stars and the planets. And after I had made copies of the books I travelled in [various] lands to seek out those nations who were expert in the wisdom of the stars and the planets,

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that I might learn from them. And I found one or two. Then I found a certain non-Jew, a wise man of Babylon whose name was Bagdas. He was schooled in the wisdom of the stars and the planets and he was able to make calculations by which he could describe the past and foretell the future. And I persuaded him by means of much wealth and many gifts to teach me the lore of the stars and the calculation of the planets.

This is practically all we possess in the way of biographical material from Donnolo himself. In the life of St. Nilus, Abbot of Rossano, Donnolo figures, however, in a very instructive incident, which throws some light on the man and his times.

On a certain day, the holy man (i.e., Nilus) came down from that place, and when he had entered the city, there approached him a certain Jew, Domnulus by name, known to him from his youth as zealous in his studies and more than commonly learned in the art of healing (*admodum studiosus et in medendi arte non vulgariter doctus*). And Domnulus addressed the abbot thus: I have heard of the ascetic way of living that you practice, and of your long abstinences, and being familiar with the condition of your body, I marvel that you have not fallen into an epileptic fit. If it please you, I will give you a medicine suitable for your constitution (*temperamento congruens*), so that you need never suffer from disease for the rest of your life. Whereupon the great Father replied: One of your own Hebrews has said to us: It is better to trust in the Lord than to trust in man. We, therefore, putting our trust in our physician God and in our Lord Jesus Christ, have no need of drugs made by hands. You will not indeed be able to impose upon the more simple-minded Christians that you have supplied Nilus with your medicines. The doctor hearing this made no reply.

While the incident has no doubt been improved for the greater glory of God and illustrates the competition that the doctors suffered from the clergy of the time, it contains at least evidence of the repute in which Donnolo was held as a physician.

The date of Donnolo's death is not known. It was after 982, so that he reached the age of at least seventy.

Donnolo's medical treatise survives only as a fragment consisting of a few leaves in a manuscript in the Mediceo-Laurenziana Library in Florence (37 Plut. 88). In 1867 the famous scholar Steinschneider edited the Hebrew text and published a German translation. The Hebrew text is full of corruptions and in many cases quite unintelligible. The treatise belongs properly to the "Antidotary" class of literature and is a fragment of a collection of recipes for the preparation of herbal medicines.

In the original MS. the treatise bears the superscription, *Sēpher hay-yākār* (The Precious Book), but is doubtful whether this formed part of the original title, as it is immediately followed by a fuller and more informing title, which I translate from the original Hebrew:—

This is the book of drugs, potions, powders, plasters, salves and unguents (?), which was composed by Shabbethai the Physician called Donnolo, the son of Abraham, who was driven out of the town Uris (i.e., Oria) in order to teach it (i.e., the book) to the physicians of Israel, and to instruct them how to prepare the drugs according to the wisdom of the physicians of Israel and Macedon, and according to the experiments he himself made with his own hands in the science of medicine, probing and plumbing the depths of medicine for forty years, by the command of God.

It is fitting (Donnolo continues) for wise and skilful physicians to have knowledge first of all of the perfumes and gums of healing plants, and of healing oils and herbs, in order to recognize them through the instruction conveyed by teachers and the books of the ancient scholars. By these means they will know and understand whether the perfumes, the juices, the oils and the herbs are pure and clean and free from any adulteration or tampering, for the sellers of these goods are deceitful and adulterate them. It will also be fitting to have knowledge of those balsams and gums which can be pounded in a mortar or sifted with a sieve, and to know what gums may be dissolved by heat, either with honey or with oil. Only then can the physician proceed to prepare such drugs as he desires.

It is also seemly for the skilful physician to know how to choose the honey which he may need for any liquid preparations; to see that it is fresh and very sweet, thick, sweet smelling, red in colour, blackish or yellowish, pure and clear.

Donnolo then goes on to discuss the various kinds of honey, singling out for special praise the Attic kind, but not forgetting the home products, the honeys of Otranto, Oria (his birthplace) and of a place in Calabria which he calls 'Martes,' which has not been identified.

If the physician, says Donnolo, wishes to mix the herb potions with honey, he must do so in this manner: After he has pounded the plants and sifted them through a very fine sieve he should warm the honey in a vessel of clay or a vessel called *Lapio*, over a fire of coals, but not upon the actual flame. He should keep on stirring the honey all the time unceasingly, in order that the honey might not boil over, and when he sees the froth come to the top, he should remove the vessel from the coals until it has cooled down somewhat. He should then remove the froth with a spoon, until all that remains is pure and clear honey. Then he should put it once again in the vessel over the coals, so that it should get hot again, and thus shed most of its moisture before it hardens and congeals. For if the honey be hard the drugs can keep for years and days without spoiling. But if the honey be moist and thin the drugs soon spoil and cannot be kept long. And after the honey has been heated according to these directions, then all the plants which have been pounded and sifted should be put in the mortar.

In Donnolo's emphasis on the medicinal value of honey we are reminded of the contemporary Anglo-Saxon herbals, where honey appears so often as a constituent of the herb potions.

Donnolo then proceeds to describe the preparation of the various herbs for medicinal purposes. Prescriptions are given for relieving constipation, the hardening of the arteries, and pain in the joints when caused by cold. The author does not tell us in what proportion these herbs should be mixed.

The fragment ends abruptly, the scribe apparently not having had any more of the original before him.

Some hundred and twenty names of medicinal plants and minerals occur in the course of this short fragment. Of these only one is definitely Arabic, two are doubtfully so; one is a literal translation of the Arabic; there are a number of Biblical and Talmudic words, and the rest are either Greek or Latin in form. I quote some names in the fragment. We find, for example, bdellium, frankincense, opium, origanum, liquid pitch, mint, ivy, hyssop, aloes, black hellebore, ammoniac, asphodel, opoponax, cedar, citron, balsam, artemisia, aristolocia, pennyroyal, wild onion, veronica, genista, tragacanth, rose-laurel, zedoary, olive, ginger and sesame.

The question arises: How far was Donnolo indebted to earlier sources and how much of the work is original? While the classical names of plants used by him are to be found mainly in Dioscorides, and a number of the Semitic terms occur in that mystery-book of Jewish medicine, *Sepher Asaph*, the actual matter found in Donnolo has not been traced to its sources. Steinschneider thinks that the book bears the stamp of an original work. And Shabbethai Donnolo more than once declares in the course of this short fragment that his prescriptions are the result of his own researches and experiments. But we must always be careful in conceding claims to originality made by mediæval authors. Considering the place and times in which Donnolo lived it can hardly be doubted that he drew his inspiration largely from Greek medical authors.

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[4] D. CASTELLI, *Il commento di Sabbatai Donnolo sul Libro della Creazione*, Florence, 1880. [5] L. VENETIANER, *Asaf Judaeus, der älteste medizinische Schriftsteller in hebräischer Sprache*, Strassburg, 2 parts, 1916 and 1917. [6] J. MÜNZ, *Die jüdischen Aerzte im Mittelalter*, Frankfort, 1922. [7] C. and D. SINGER, "The Origin of the Medical School of Salerno" in *Essays on the History of Medicine presented to K. Sudhoff*, edited by C. Singer and H. E. Sigerist, Oxford, 1924.

[*February* 1, 1928.]

The Gild of Barber Surgeons of the City of York.

By G. A. AUDEN, M.D., F.R.C.P.

THE origin of the Gild of Barber Surgeons of the City of York cannot be traced, but is of great antiquity. The Roll of Freemen of the city begins in 1272, and the entry of the first barber is in 1299, when occur the names of Adam de Stokton and Rogerus le barber. Three years later comes Thomas, son of Hugo le barber. It is probable, therefore, that at this date there was some barbers' organization recognized by the city authorities. In the next fifty years seventeen barbers were admitted. Two at least attained a high reputation in their profession, as is testified in the following entry in the accounts of John Wodehouse, Clerk to the Exchequer, under the date 1346 (20 and 21 Edward III): "Payments to William de Bolton and Hugo de Kilvington, barber-surgeons, going from York to the Castle of Bamburgh to heal the said David de Brus who lay there, having been wounded with an arrow at the said battle (*Neville's Cross*), and to extract the arrow and to heal him with dispatch, £6."[1] Their treatment was successful, for David Bruce, King of Scotland, lived for many years afterwards.

The will of another barber surgeon of this period, Ricardus de Dalton (freedom 1350), is printed in the *Testamenta Eboracensia* of the Surtees Society.

In the Corporation House Book, under January 23, 1393-4, is a curious reference to the means whereby one barber surgeon protected himself against a possible charge of malfeisance before undertaking an operation on a colleague.

Venerunt in camera maioris super pontem Use in Eboraco coram Johanne de Brathwayt, tunc maiore, Alicia, uxor Johannis de Cartmell, barbour (*freedom 1391*). Robertus Talke tunc camerarius, Johannes de Kilvyngton, Hugo Gardyner, Johannes de Man, tapiter, et alii vicini ipsius Johannes de Cartmell, testificantes quod quia ipse Johannes nimis languidus fuerat de quadam infirmitate vocata le stane, peciit et requirebat quod Johannes Catlewe (Roll of Freemen gives Cattlogh: *freedom 1388*) de Ebor; barbour, curam sibi imponere vellet ad afferendam petram; et sive viveret sive moreretur, ipse Johannes Catlewe quietus fore deberet ab omni impeticione, calumpnia et demanda per ipsum seu dictam Aliciam uxorem suam, aut quemcumque alium aliqualiter in hac parte facienda.

The earliest ordinances preserved are contained in the Corporation House Books. These, which are of about 1400, show the Gild as governed by elected officers. They point to its originally religious character, for they refer to the light to be maintained by the Gild, and to the Pageant in which the Gild took its part. This combination for religious observance for the mutual support and for regulation of professional conduct, was the origin of the similar gilds of Barber Surgeons in London, Lincoln and Norwich.

The pageant (pagina) was probably that of the Corpus Christi. The Gild of Corpus Christi became a very popular brotherhood, to which belonged many

1 Quoted by Purey Cust, *Heraldry of York Minster*, vol. ii, p. 404. The arms of Bolton are exhibited immediately above the N.E. arch of the Nave of the Minster, bearing on a chevron three lions passant guardant. Perhaps these arms were granted as a reward for services rendered on this occasion.

[2] $\frac{A}{Y}$ folio LXXija.

prominent citizens and their wives. There are no barber surgeons in the lists of members, probably due to the religious character of their own Gild. Several apothecaries, however, appear in the lists, e.g., John Browneflete (freedom 1430), who was Sheriff of the City in 1426, William Rukshawe (Rowkshawe), who was Chamberlain in 1460, and George Essex (freedom) 1473, who was fined ten marks for some reason in 1510. The Gild was suppressed in 1547.[1]

The York Craft Gilds took part in the plays acted in the streets of York at the Festival of Corpus Christi, and the House Book gives a list of these plays for 1415. That of the Barbers was the Baptism of Jesus by S. John Baptist. These plays are in a manuscript now in the British Museum. (Additional manuscript 35290.) They have been edited by Miss L. Toulmin Smith.

The early ordinances of the Gild of Barber Surgeons of York have been published by Dr. M. Sellers (*Surtees Society*). It is therefore unnecessary to reprint them here. There is, however, a curious inclusion of certain of these ordinances in those of the Goldsmiths.[2] It runs as follows :

Item que ceaux que sount aleins ou autres estraunges usent fisyk ou surgerie deincz la citee et preignent avauntage pur lours affaires, qils soient contributours a sustener et mayntener lour dite et lour lumer sur peyne de vj s. viij d. a paier en la fourme suisdite.

Item si ascun soit rebel du dit artifice, pur venir a lour assemble, pur ordeigner necessarie purveaunce pur lour pagyne lumer our autre ordeignaunce lisible et honest affaire par convenable garnishment, sinon qil est resonable excusacion il paiera une livere de cire a lour lumer.

Item, si ascun mestre preigne ou coille autre servaunt ou apprentice du dit artifice avant qil od servi son dit mestre pur le terme accorde entre eaux, il paiera vj s. viij d. en maner come desuis et ditz.

The prohibition of Sunday labour fell into a certain measure of disuse. In 1413, Thomas Arundel, Archbishop of Canterbury, previously Archbishop of York, addressed a letter to the Mayor and Aldermen of London.

Sons in Christ and dearest friends—we know that ye do seek for the things that are above and that ye will the more readily incline to our desires, the more surely that the things as to which we write are known to tend to the observance of the Divine law, the maintenance of public propriety and the rule of the christian profession. We do, therefore, write unto you on this occasion to intimate that when we were presiding of late in our Provincial Council holden in London . . . . it was publicly made known unto us with universal reprobation that the barbers of the City of London . . . . in their blindness do keep their houses and shops patent and open on the seventh day—the Lord's Day namely—and do follow their craft on the same . . . . Wherefore we, ... in restraint of such temerity as this, have determined that there must be made solemn prohibition thereof in the city aforesaid . . . . that such barbers must not keep their houses and shops patent and open, or follow their craft, on such Lord's Days for the future, on pain of the greater excommunication, in the same manner as it has been enacted and observed of late in our time as to the City and Diocese of York, as we do well recollect.

Written at Ikham[3] on the 13th day of the month of July (1413).

Thomas, Archbishop of Canterbury.

Immediately following the Latin Ordinances in the House Book are a series of additional ordinances, in English, relating to practice by aliens and strangers. These can be dated as 1485 :

Statutes and ordinances maid and addid unto the Ordinances of Barbours at their grete instance and labours.

ffirst that the Searchours of the Barbours of the Citie of York for the tyme being shall, at all tymes have powre to serch all and everie Aliene and Straunger comying to this Citie to

[1] Skaife, Surtees Society, 1874. *Gild of Corpus Christi.*

[2] Sellers. Surtees Society. *York Memorandum Books.* Vol. i, p. 75.

[3] Letter Book i, 125 (Ikham now Ickham, near Wingham, Kent).

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exercise any poynt of surgerie within ye same. Also, them foundon able and admitte by the sad serchers to exercise the craft of surgerie or other, pay incontinent upon their admission to the Chambre and the Craft equally, vjs. viijd.

Also that all suche Aliens and Straungers foundon without a faut by the said Serchours in the said Craft of Surgerie be finable and reformed according to ye ordinance and statutes made in the same craft.

Also that ye Serchours of the Craft of Barbours foresaid for ye tyme being shal have power in all tymes as they shall seme to serch all maner cures of surgerie which the said Aliens and Straungers shal have in hand of and inhabiting within the said Citie, suburbs and precincts of ye same.

At this time the rules and ordinances of the Gild were revised. The Book of Ordinances, which is now preserved in the British Museum (Egerton MSS. 2572) was begun in the following year. This book has been edited in full by the late Dr. Furnivall in the Early English Text Society, LIII, 1888.

We are thus fortunate in having an unusually complete series of the additional rules, which were added from time to time, and are able to follow closely the growth and activities of the Gild until it fell into decay and final dissolution. The last entry in the book runs: "This book repaired June 10, 1774, by Mr. Richard Hardy [freedom 1756], and Mr. Wm. Harrison [freedom 1768], searchers to the Barber Chirurgeons." "William Bratherton, an unfreeman's son, bound Feb. 1, 1781."[1]

This book in the British Museum does not contain the ordinances which have been transcribed by the Surtees Society from the Corporation House Books, and it would appear that a number of pages were left blank with the intention of inserting them. In their place, however, was added a painting of the coat of arms of the Barber Surgeons and portraits of Henry VII, Henry VIII, Edward VI, Queen Mary, Elizabeth, James I and Charles I. Then follows an entry to which a marginal note to the Latin ordinances makes references, viz., the statement:

The auntiente ordinarye of the Barbors and Surgions of this Cittie, att the requeste of the wholl companye, newlye perused, reformed and Augmented, and this presente XXI daye of June, 1592, ratyfied, established and confirmed, to be henceforthe observed and kept, as hereafter mencyoned.

These ordinances are purely administrative and for the most part expansions of the earlier ordinances. All reference to the light and pageant is omitted, for the Gild of Corpus Christi ended with the Reformation. The Monday following the Feast of the Nativity of St. John Baptist is retained as the day for the annual election of the officers and for the passing of the accounts. Barbers are allowed a little more latitude in the matter of Sunday observance and are permitted to open their shops on the two Sundays immediately preceding the Assizes. No one may practise surgery or dental extraction, except supervised by a master. All strangers practising as physicians or surgeons must contribute 6s. 8d. per annum to the Gild and their work is to be supervised by appointed officers. There is an interesting variant reading of this item in the Corporation House Book copy. In the Egerton MS. it runs: "item that no person or persons within this Cittie, or suburbes of the same, practising Chierurgerye": but in the Guildhall MS this reads: "That no man or woman within this Cittye." We have, moreover, evidence of the practice of surgery by women in an entry in the House Book under the date 3rd June, 1572:—

forasmoche as it apereth that Isabell Warwike hath skill in the Scyens of Surgery and hath done good therein; it is therefor aggreed by they present that she upon her good behavor shall use the same sciens within this Citie without lett of any of the Surgians of the same.

No member of the Company might intrude himself into the practice of another member, except by request of the patient or his friends, nor undertake to "powle

[1] William Bratherton did not take his freedom of the City.

trim or shave any of his brother's customers untill suche tyme as the said brother be fullie contented and paide."

The last three items of the Egerton MS. do not appear in the York Guildhall Manuscript. These (*a*) prohibit the admission of a brother to the Company before he has received his freedom, (*b*) prohibit the taking of more than one apprentice at a time, and (*c*) ordain that the apprentice must always be the son of a freeman.

That the searchers seem to have occasionally met with difficulty in enforcing their ruling on surgeons who were not members of the Company is indicated by an entry in the House Book. Thus, under July 3, 1598:—

William Padmore, surgeon, was complained on by ye company for exercising chirurgery in this cittie; not being a Freeman, who said for excuse yt he was allowed and licensed to practice by my Lord's Grace under his seal; but ye Court did not think him to be excused thereby, but ordered him to pay penalty in ye Surgeons' ordinarie, which is two shillings per diem soe long as he practised, unless he can quit himself by law: and upon the 28th July, 1598, he refused either to pay ye said penalty or to take his freedome; whereupon he was comitted till he paid ye said penalty, and on the 20th October, 1598, it was agreed yt William Padmore shall be made free for £6 13. 4.

Occasionally the Council seems to have overridden the objections of the Company by admitting to practice one who did not conform to the conditions of these ordinances. Thus under September 12, 1608: "John Grai, a travilor, a chirurgeon, being skilful, namelie esspeciallie as he saieth in fowre diseases" who claimed that he was able to help "those that have wanted their sights this twentie yeares past within neyne daies to recover their sight" was licensed to practice his art within the city.[1]

The London Corporation of Barber Surgeons in 1555 resolved to nominate two of their members to give instruction in anatomy to the general body. Possibly some such instruction was given in York, but it was not until 1614 that new ordinances were passed to place it on a definite footing. It was then enacted that

the companye of chirurgions [note the separation from the Barbers, who are always co-named in the previous ordinances] "everie yeare shall choise one of the saide companye to be the Maister in Anatomie."

The Master so chosen must be a licensed surgeon and twice during his year of office he was under obligation to read a lecture in anatomy or surgery, in default he must pay a fine. Every member of the company must attend the dissections under a penalty of 3 shillings and 4d. for absence.

The master could name "such of the licensed chirurgions as he shall like best of" as Prosectors under his supervision and "shall describe to such as he shall appoint to dissect (if they be unskillfull in ye dissection of that part) the rising, circumference, site and insertion of the said part: which if he do not, they requestinge him thereunto, he shall pay iijs. iiijd. to thuse aforesaide."

Every surgeon in York was compelled to join the Company or be expelled from the city, and within a month of being made a freeman had to read a lecture on Anatomy or Surgery, and every member of the Company was under the obligation of reading "to the whole companye, out of some Aucthor in Chirurgerye or Anatomye, as shalbe appointed by the Maister in Anatomy."

On the other hand, every freeman or woman of the city was compelled to consult a licensed surgeon under a penalty of 40/- before consulting any unlicensed practitioner, who were rigorously dealt with by the searchers.

None unlicenced, or suche as can give no reason for the cure they undertake, as to have knowledge of the causes and signs thereof, or none that understande not the vertues of such medicines as they applie; whether theye be simple or compounde, takinge moneye for these

[1] M. Sellers, Victoria County History, *Yorkshire*, p. 470.

medicines, shall practize Chirurgerie upon payne to forfeit for everye tyme they shall practize Chirurgerie within this Cittie xxs.

The Company had soon occasion to put this last ordinance into practice, for in the list of Masters and Apprentices appended to the Egerton MS. is the entry: "Francis Brigham, Mr. but insufficiente for Surgerie, and also againe the XXth Aprill 1615 he is found insufficiente with full approval and consent of the whole companye."

The result is known to us from the Corporation House Book under date June 21, 1615:—

Upon 8te. Maye by the Barber Chirurgeons against Frances Brigham, who professeth Himself to be a Barber Chirurgeon, of his insufficiency in the said art, which they manifested. It was thought mete and so ordred by this Court by consent of the saide parties that the said Francis Brigham should not meddle with any cure unlesse he be assisted by a skilfull and sufficiente Chirurgeon lawfully licensed.

The appointment of a Master of Anatomy and the institution of systematic dissections necessarily raised the question of the supply of bodies. On this the Corporation House Book has the following entry:—

And the following articles were also ordered to be added to the Barber-Chirurgeon's ordinary at a meeting of the Corporation held on the September 8, 1615.

1. That the Maister of Anatomie at everie assizes, if they can procure or gett a bodie executed at everie the Assizes, cause it to be brought to some convenient place to be dissected, and in caise he fail therein to forfait for everie such defalt xxs, to thuse of the Maior and Commonalty of this Cittye.

2. That the Maister of Anatomye and one of the Searchers, being a licensed Chirurgeon shall once in six weeks at the least, not being on a markett daie, cause the apprentices of suche as practize Chirurgerie to mete, and to examyn them upon suche pointes of Chirurgerie as they shall think mete for the betteringe of their knowledge therein. And those that shall faile to send their apprentices te be examined, not havinge a reasonable excuse to forfait for everie defalt iijs. iijd. to thuse aforesaide. item. All that practize Chirurgerie shall yearlie contribute toward the chardge of the anatomie proporcionally according to their several estats and sufficiencies, and they shal be present at the same and learne to dissect upon payne of everye refuseing or makeing defalt to pay iijs. iiijd. to the use aforesaide.

But the appointment of a Master of Anatomy led to another difficulty, and quickly introduced an element of discord into the fraternal gatherings of the Company. There was dispute as to precedence between the master and the searchers. This was finally settled by the mayor and aldermen, in 1614, in favour of the master (Egerton MS.).

The later additions to the ordinances need not detain us. They appear in the Early English Text Society's transcription, and refer for the most part to Sunday trading, to finances and to the imposition of fines. There is, however, one entry which does not appear in that transcription, dated May 6, 1701, which is accordingly given here:—

Ordered and agreed by the company att a Hall held this day that every searcher of the company for the time being shall hereafter, before he spends the stock and other things belonging to the company, enter into bond with sufficient sureties at the discretion of the ffeoffees for the time being not to expend or otherwise waste any of the stock belonging to the company except two shillings and sixpence upon every quarter day, and what other expends. upon any other account shall be ordered and allowed by both or one of the said ffeoffees for the time being, and the said ffeoffees or one of them to be present all or order all such other expends. as shall for the future be expended by the searchers for the time being, otherwise not to be allowed off in their accounts.

Some twenty-five years previously it had been enacted that the searchers should not spend money or stock belonging to the Company "in ffeasting or in any other way." In 1679 the searchers became entitled, on payment of 10s. yearly to the

mayor, to receive all "fines and forfeitures due by breach of any Artickle of this ordinary: fines due or forfeitures taken of Doctours or Montebankes only excepted."

Apart from the Barber-Surgeons, who received their freedom of the city, the Freemen's Roll has many names of those who practised medicine and physic in contradistinction to surgery. They were probably in many cases men of better education and social standing than the barbers, and in some cases graduated at universities. In some instances they appear to have been foreigners. Thus we

Searcher's Badge. York Barber-Surgeons (mounted on an oak frame) now in the possession of the Wellcome Historical Medical Museum.[1]

find "Mr. Johannes de Golda Miles in medicinis" (1396), "Johannes, fil. Hugo de Metice, Medicus" (1398), and "Graciamus de Agnero in Hispania, oriundus medicus" (1502), and much later "Doctor de Montanibus" (1680). Whether the designation "phisicus," which first appears in 1419, had any special connotation is doubtful, or the term "leche," by which Adam de Fendyk is so designated in 1375. In 1402 there appears "Duncanus Leche" without further designation. It is, however, interesting as evidence of the hereditary character of the calling to find in 1536 Christofer Leche, the forerunner of eleven members of seven successive generations of that name who became members of the Barber Surgeons Company, the last being Joseph Leech, who received his freedom as

[1] Copyright of the Wellcome Historical Medical Museum, reproduced by permission. Another Searcher's Badge of the Company is now in the possession of W. A. Evelyn, Esq., M.D., York.

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Barber Chirurgion in 1721. During the eighteenth century membership of the company ceased to be obligatory upon surgeons practising in York. Thus Francis Drake, the author of *Eboracum* and surgeon to the County Hospital, who was the "City Surgeon" until dismissed on his refusal to take the oath of fealty to the Government in 1745, does not appear to have received his freedom, or to have been a member of the Company. This also applies to Dr. John Burton, the author of *Monasticon Eboracense*, and prototype of Laurence Sterne's "Dr. Slop" in *Tristram Shandy*, who practised for many years in York as man-midwife and physician.

[*March* 7, 1928.]

A Medical Adventurer. Biographical Note on Sir James Wylie, Bart., M.D., 1758 to 1854.

By Robert Hutchison, M.D., F.R.C.P.

Until well on in the nineteenth century there was almost no organized profession of medicine in Russia. Treatment amongst the lower orders was still largely in the hands of the clergy, whilst for the court and nobility medical talent was derived from abroad. Many of the imported doctors were Scots,[1] driven by the poverty of their country and the spirit of adventure to seek their living abroad. Of these "doctors of fortune" James Wylie was a conspicuous example.[2]

He was born in Kincardine in 1768 of humble parentage, studied medicine at Edinburgh, and afterwards at Aberdeen, taking the degree of M.D. there in 1794. Soon after he entered the Russian Medical Service. About this time the Tzar Paul had ennobled one of his lower attendants. Wylie was fortunate enough to treat this Count Kutaisoff successfully for an abscess in the throat, which he incised, after several Court doctors had failed to cure him. He was at once raised by the Tzar to the position of Court Physician.

Other cases, equally fortunate for Wylie, occurred soon afterwards, so that, as Lyall says, he might in truth be regarded "as having risen upon the ignorance or stupidity of his contemporaries." His position, however, remained very insecure, for his good fortune caused much jealousy.

Paul was assassinated in 1801. He was attacked in his room in the palace by four or five officers. After a desperate struggle he was stunned by a blow on the head from the butt end of a pistol and finally strangled with a scarf. Wylie embalmed the body of the Emperor, and D. de Joyneville tells us that he signed a certificate that he died from the effects of apoplexy. He adds that "Dr. Wylie also thought that, allowing for the injuries caused by the mode of death, the state of the brain rendered it highly probable that the Emperor would soon have died of apoplexy"!

Wylie became a favourite of Paul's successor Alexander, and accompanied him on all his travels. He was present with him at the battle of Borodino and performed 200 operations on the field. He also witnessed with the Tzar the scenes at Wilna in 1812. During the retreat from Moscow he became possessed of a bit of loot in the shape of a pair of pistols, still in possession of my family, and which, according to

1 Amongst these were Erskine (a relation of the Earl of Mar), Calderwood, Matthew Guthrie, Robert Simpson, Charles Brown, and Samuel Hurst. Dr. John Rogerson, an Edinburgh graduate and native of Dumfries-shire, also went to Russia in 1766 and remained there for fifty years, during most of which period he was body physician to the Empress Catherine and her successors. (*Lancet*, August 7, 1897.)

2 I confess to having always taken a special interest in the career of Sir James Wylie, as he is my one medical ancestor. He was an uncle of my paternal grandmother.—R. H.

a family tradition, were taken from Napoleon's carriage. At Dresden, in 1813, he amputated Moreau's legs, which were shattered by a cannon shot as he was talking to the Tzar.

Wylie accompanied Tzar Alexander in his last journey to the Crimea. There his biographer, Dr. Robert Lee, who was in the service of Count Woronzow, met him. During the journey Alexander appears to have contracted the malaria, which led to his death in the following year (1825). Wylie, along with other physicians, attended him in his last illness, but Alexander declined to take any of their prescriptions. After his death the physicians were accused of mismanaging the case. Lee handsomely exonerates Wylie from all blame. "I enjoyed," he writes, "the best opportunities in the Crimea of observing the devoted attachment of Sir James Wylie to the Emperor Alexander, whom he had accompanied on all his campaigns, and I conscientiously believe that on this trying occasion Sir James discharged his arduous professional duties in a manner worthy of his high reputation."

Alexander was succeeded by Nicholas, but Wylie kept his place and became as much a favourite with the new Emperor as with Alexander, attending him everywhere. He continued in the full sunshine of royal favour and worldly prosperity until his death, which took place in St. Petersburg in 1854 at the age of 86.

A few words as to his character and ability. Lyall, who wrote during his lifetime, says that he "is one of the most notorious and powerful individuals in Russia. . . . With not very brilliant medical talents, with but very moderate scientific acquirements and with much singularity and little refinement of manners, Sir James Wylie has risen from the most obscure parentage to be the first medical person in the Russian Empire." Of Wylie's medical knowledge there are no grounds on which to form an estimate. He wrote a few medical papers, some in Russian and some in French, chiefly on yellow fever and plague. With the assistance of a number of professors he composed a pharmacopœia of which Lyall remarks that it "has its uses in Russia, though a work of little ingenuity or merit, and unfortunately the knight in his ardour for the adoption of new names has composed some almost the width of a page, than which nothing can be more ridiculous." Another instance of this "ardour" seems to have been his invention of the curious term "cardiopalmus" to dénote palpitation of the heart and great vessels, so common in the Russian army.

Lee thought that Wylie had leanings to homœopathy, but apparently on no stronger grounds than that at a social discussion he shrewdly said that "Hahnemann with his minute doses cured as many patients as regular physicians did by their great ones, because he at the same time enjoined a rigorous diet." Lyall says that he was reckoned a good surgeon and an expert lithotomist. He had ample surgical experience in the field. He was certainly a capable administrator. As early as 1800 he took a foremost part in founding the Medico-Chirurgical Academy at St. Petersburg and in 1812 he was made Director of the Medical Department of the Ministry of War. Lyall admits that in this capacity he was "most assiduous in his duties and introduced numerous improvements into surgical practice and reorganized the military hospitals." Lefevre writes that: "It is to Sir James Wylie that Russia is indebted for the organization of her medical schools, both civil and military. . . . The common soldier has to thank him for such care and protection as his predecessor in arms demanded in vain and the army in general has to thank him for a real and effective instead of an inefficient staff."

Wylie was eccentric, and possibly a little conceited. Lyall says: "He has acquired much importance in affairs not belonging to his department. Princes, Generals, and Officers of all ranks are daily seen at his levees, some of whom he treats as if they were quite his dependants. But as he has obtained a character for

oddity and drollery . . . every liberty is excused. He lives in the Imperial Palace in the most niggardly manner and seldom or never pays for a dinner. If obliged to remain at home, soldier-like he makes his repast on black-bread and salt. But in general he goes, without invitation, to some acquaintance either in the Palace or the City and dines *en famille* agreeably to the custom of the country."

Wylie received many honours. He was knighted in 1814 by the Prince Regent, some say on board a Russian warship off Portsmouth; others on Ascot Heath. In view of the Prince Regent's habits the latter seems the more likely. The sword of the Cossack leader Platof is said to have been used on the occasion. In the same year he was made a Baronet of the United Kingdom by special request of the Tzar. In Russia he was not only Body Surgeon and Physician to the Tzar, Chief of the Military Medical Department and President of the Medico-Chirurgical Academy, but held the office of Privy Councillor and the Russian Orders of St. Wladimir, St. Alexander Newsky, and St. Anne, as well as the foreign decorations of Legion of Honour, the Red Eagle of Prussia, the Crown of Würtemburg and the Leopold of Austria. He is shown wearing the insignia of some of these orders in this portrait, which is a replica of one in the possession of my family. He was also elected a member of almost all the learned societies in Russia, as well as of some in Great Britain and upon the Continent.

Although he did not undertake private practice. Wylie enjoyed the revenues of two estates conferred upon him by the Emperor and amassed a fortune. Being without heir he bequeathed it to the Tzar, but his Scotch relations succeeded, after some litigation, in recovering a considerable sum which had been invested in England. To do his Royal master justice, he did not keep Wylie's money, but used it to found a military hospital, which is said to have cost £100,000, and which was opened in 1873. It was called "The Michael Clinical Hospital of Baronet Wylie," and occupied three sides of a square, in the centre of which was a life-size statute of Sir James. It stood, and, so far as I know, still stands in the city now known as Leningrad.

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Clinical Section.

President—Sir HERBERT WATERHOUSE, F.R.C.S.

[*May* 11, 1928.]

A Case of Post-operative Tetany.

By DONALD HUNTER, M.D.

G. B., FEMALE, aged 44 years.

History.—Patient was born at Wolverhampton and lived there till the age of 21. Goitre was certainly endemic there, though no other member of her family was affected. At the age of 18 she first noticed a small swelling in the right side of her neck. This gradually enlarged and extended to the middle line. At the age of 22 the swelling rapidly increased, became painful and tense, and caused dyspnœa and hoarseness. She was admitted to the Queen's Hospital, Birmingham, where the right lobe and isthmus of the thyroid gland were found to be greatly enlarged, hard and tense. The left lobe was firm and enlarged to a lesser degree. The diagnosis recorded in the hospital notes is "bronchocele."

On September 19, 1906, subtotal thyroidectomy was performed by Mr. Jordan Lloyd, only a portion of the isthmus of the gland being left. Severe lateral compression of the trachea by the tumour was found. After it was removed the recurrent laryngeal nerves were seen intact on both sides of the neck.

Primary union occurred and the patient was discharged well two weeks after the operation. According to her statement the tumour weighed a pound and a half. There is no record of any histological examination.

About two weeks later, when she was beginning to get about, she noticed that she could not use her hands properly because they both "drew up." In such attacks the fingers tingled, felt to some extent numb and became stiff to such a degree that the thumb was pressed against the middle and ring fingers causing pain. Sometimes there was pain also in the dorsum of the hand and in the forearm. Stiffness of the forearms and flexion at the wrists and elbows occasionally occurred. The symptoms could be brought on by raising the arms above the head. During bad attacks the feet and legs were affected so that she found it difficult to sit comfortably. The feet felt stiff and were turned inwards at the ankles, the toes being "drawn under the foot."

During the first four years after operation the stiffness in the hands would often persist for many days together. The skin became slightly dry, but the hair and nails were unaffected and there was no mental dullness. Before the operation she could sing well, but she has been unable since to reach the high notes. During the third year after operation she was treated with thyroid extract, 5 gr. daily. This treatment has been repeated intermittently during the past two years, but on no occasion has it seemed to influence the attacks of tetany. She has never been advised as to a special diet and has never been given calcium salts.

Four years after operation the attacks spontaneously disappeared and she had no symptoms for six years. Then, in 1916, when it was particularly difficult to get food owing to the war, the attacks returned and became more severe. Recently she has had attacks every four weeks, sometimes every day for a whole week. She has been continuously worried by spontaneous twitchings of the limbs and

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fingers, and at night she has been unable to lie still. The fingers, wrists and elbows have rarely been free from discomfort.

Two years ago she began to have attacks in which peculiar ocular phenomena occurred. These were worst when the hands were very bad. They often persisted two days and recurred perhaps once in four weeks. Apparently on extreme lateral deviation of the eyes to either side she was unable to bring them back to the resting posture for several seconds. In addition she found that after reading or doing needlework she experienced dimness of vision and giddiness on looking at distant objects.

In the four weeks immediately preceding her admission to the London Hospital she had attacks of tetany worse than ever before. In these attacks the carpo-pedal spasm was accompanied by spasm of the facial muscles, so that the face and lips were "drawn tight and aching." At such times she found it difficult to open her mouth to talk or eat. Sometimes there was spasm of the laryngeal and pharyngeal muscles so that for a few minutes she could neither speak nor swallow. No attack was accompanied by convulsions.

Past History.—The patient is a single woman who keeps house for her mother and brother. She has two brothers and one sister alive and well. Her father died of pulmonary tuberculosis. The catamenia commenced at fourteen years; they are normal, perfectly regular, and last three to five days; the operation in no way affected them. She has had no other illness of consequence. The teeth apparently were erupted in the normal way. In 1912 she broke a tooth while eating; the upper jaw was cleared and an upper denture made. In 1919 more teeth were removed and a lower denture made. In the same year the refraction was tested and she was ordered glasses for reading and sewing.

On Examination.—A woman of slender build. Height, 5 ft. 2½ in. Weight, 7 st. 3 lb. Intelligence, memory, and mentality normal. Mucous membranes normal in appearance. Skin of good colour and normal texture. Finger-nails and toe-nails healthy, with lunules and longitudinal striations. Hair of scalp, eyebrows, eyelashes, pubes and axillæ normal. The eight remaining teeth appear to be normally calcified. Dental caries and gingivitis present. Voice and hearing normal. Examination of larynx negative. Pupils, equal, react to light and on convergence. Ocular movements full. Visual acuity good. Slight error of refraction. Fundi negative.

Examination of the lens (fig. 1) with the slit lamp (Mr. Goulden):—

Very superficial white flaky opacities near the anterior and posterior capsule of the lens. A few red, blue, and green crystalline opacities also. A few powdery white opacities in the nucleus.

Extensive "collar" scar of thyroidectomy in the neck. Thyroid gland not palpable: rings of trachea easily felt. No accessory thyroid palpable in neck.

Twitchings and tremor of face with pursing of lips in talking ("carp mouth"). Light percussion over all the branches of the facial nerve induces twitching of the face (Chvostek sign).

Inflation of a sphygmomanometer bag on the upper arm produces typical tetany in thirty seconds. There is flexion at the wrist and metacarpo-phalangeal joints, with extension of the inter-phalangeal joints, the thumb being pressed against the third finger (fig. 2). Severe discomfort is caused by the test (Trousseau sign).

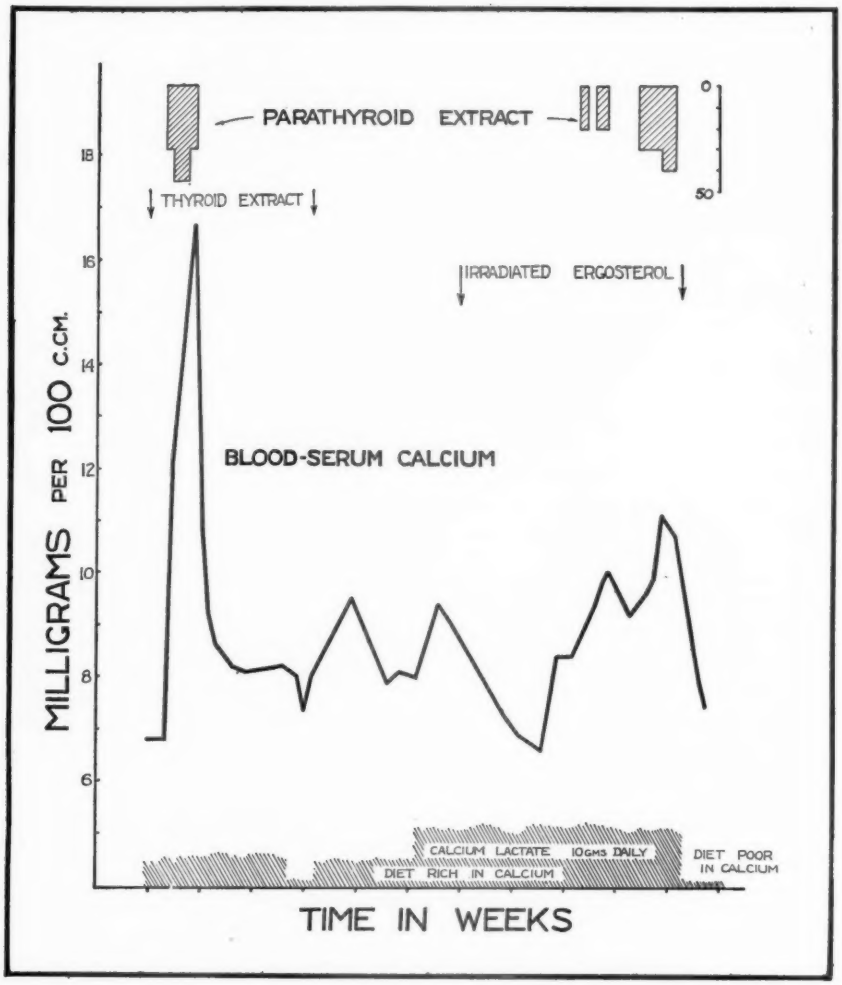
There is increased electrical excitability of the muscles. Thus, for example a brisk contraction of the palmaris longus is obtained with a current (K.O.C.) of less than 2 milliampères (Erb sign).

Examination of heart, lungs and abdomen is negative. No weakness nor wasting detected in muscles of limbs or elsewhere. Tendon reflexes normal, plantar responses

FIG. 1

FIG. 2.

flexor. Blood-pressure 130/80. Urine and renal efficiency tests normal. Wassermann reaction in blood negative. Blood-count normal. Serum calcium 6·8 mgm. per 100 c.c. Inorganic phosphate in serum 5·4 mgm. per 100 c.c. Basal metabolic rate – 5 per cent.



Radiograms show no evidence of decrease in calcium salts in the bones or teeth.

Treatment.—This is set out in the Chart in such a way as to indicate variations in the serum calcium. The period covered is approximately twelve weeks. The patient was first given a diet rich in calcium salts. The calcium content of this

diet was not estimated; she was simply given as much milk and eggs as she could take. Collip's parathyroid extract (Eli Lilly) was given by intramuscular injection, starting with 10 units and rising to 15 units three times a day. After 150 units had been given the serum calcium had risen to 16·6 mgm. per 100 c.c. It should be noted that during the first four weeks of treatment thyroid extract (6 gr. daily) was given by mouth.

As soon as the serum calcium rose to normal the patient stated that she had completely lost all her symptoms for the first time in twelve years, and felt better than she had ever done since the operation. On physical examination restlessness and spontaneous twitchings were absent, and the Trousseau, Chvostek and Erb signs had disappeared.

In the fourth week she was placed on a diet poor in calcium; that is to say milk and eggs in all forms were omitted. All the signs and symptoms of tetany returned, the serum calcium dropping to 7·4 mgm. per 100 c.c. The resumption of a high calcium diet then abolished the symptoms, and the serum calcium showed a temporary rise to 9·5 mgm. per 100 c.c. Addition of calcium lactate to this diet caused a second temporary rise in the serum calcium. It was given in doses of 5 grm. twice daily before meals. Irradiated ergosterol (British Drug Houses) was given in pellets (two daily) over a period of four weeks. It was apparently without effect on the serum calcium.

During this time, although the calcium level in the serum once fell as low as 6·6 mgm. per 100 c.c., no symptoms nor signs of tetany appeared. Later, parathyroid extract was again given and the serum calcium rapidly rose to the normal level.

Comment.—The symptoms and signs of parathyroid deficiency in this case are very characteristic. Those of thyroid deficiency are curiously lacking.

The occurrence of cataract in post-operative tetany is well known. Thus Sainton and Péron [1] describe one case and refer to six others, while Aub [2] has since published a further case. Erdheim [3] produced cataract by experimental parathyroidectomy in rats. With the slit-lamp very early opacities can be detected in the lens, and Knüsel [4] has described four such cases, all occurring in patients with post-operative tetany. Mr. Goulden [5] kindly examined the present case, the slit-lamp appearances of which he is publishing elsewhere.

The rational treatment of tetania parathyreopriva is to supply the calcium mobilizing hormone [6]. Only the less severe cases, such as that described above, react to treatment with calcium salts by mouth, and it must be recognized that the response of the serum calcium to such measures is very limited [7]. In all severe cases the patient should be given a high calcium diet, large doses of calcium lactate by mouth and injections of parathyroid extract. Hypercalcæmia, though it causes no harm if not protracted [8], should be avoided. There is unfortunately evidence [9] that after many months of repeated use parathyroid extract sometimes fails to raise the serum calcium. The exact reasons for this have not yet been elucidated.

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Discussion.—Sir HERBERT WATERHOUSE (President) asked if there were any statistics showing how frequently thyroidectomy was followed by tetany. He, personally, had not met with such a sequel. Was it not likely that at the operation on this patient the parathyroids had also been removed?

100 Hunter—Cassidy and Max Page: *Carotid Body Tumour*

Dr. F. PARKES WEBER said he had only once seen spontaneous "idiopathic" tetany in an adult—a young woman, many years ago. He would, however, like to know whether the modern method of treatment for post-operative tetany, described by Dr. Hunter, was likely to be useful also in the very rare spontaneous cases in adults.

Dr. H. STANNUS asked what the subsequent treatment of this patient should be? Would the injections have to be continued for the rest of the patient's life?

Dr. DONALD HUNTER (in reply) said it was striking that post-operative tetany should be so rare. It was not so in all countries, especially in those where operations for endemic goitre were common. Thus Knüsel, working in Switzerland, was able to find four cases at one time. In the United States, where it was customary to remove nine-tenths of the thyroid gland in exophthalmic goitre, post-operative tetany was more common than in this country. Some American surgeons searched the tissue removed at operation for parathyroid glands in order that these might be at once transplanted. Lahey, of the Deaconess Hospital, Boston, examined microscopically such autotransplants and found parathyroid tissue in only 5 per cent. The fact that radical thyroidectomy was not a common practice in this country might explain the rarity of post-operative tetany.

He had had no experience of "idiopathic" tetany. In cases of tetany associated with rickets, the symptoms and signs of tetany disappeared on treatment with Collip's parathyroid extract, though of course the rational procedure was to treat the rickets.

The patient under discussion had improved very much on calcium lactate, given in addition to a diet rich in calcium salts. Therefore, unless she had tetany again, he intended to discontinue the use of parathyroid extract. But he had seen a case of post-operative tetany where the serum calcium had been 4·3 mgm. per 100 c.c., and here, in addition to a diet rich in calcium salts, it was imperative to use injections of an active parathyroid extract and to continue them indefinitely.

Case of Carotid Body Tumour with Syncopal Attacks, after Operation.

By MAURICE CASSIDY, M.D., and C. MAX PAGE, D.S.O., M.S.

THIS patient was shown at the February meeting of the Section. He had six syncopal attacks during the past four years and had noticed a lump in the left side of the neck one year ago. Pressure on this lump produced slowing or stopping of the

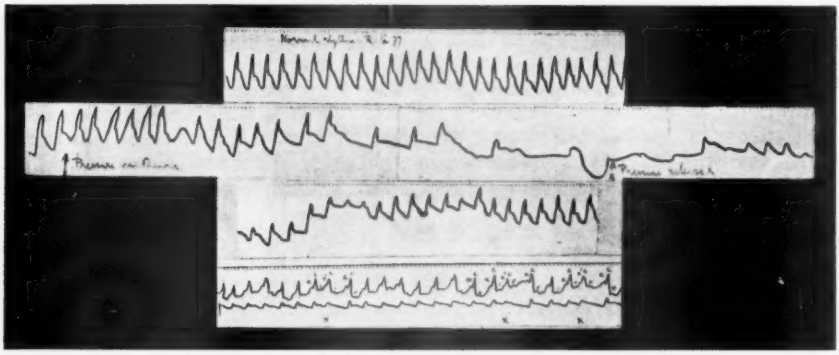

FIG. 1.—Upper three strips are continuous radial tracings showing effect of pressure on tumour, slowing and stopping radial pulse. Lowest strip shows arrhythmia due to premature auricular beats.

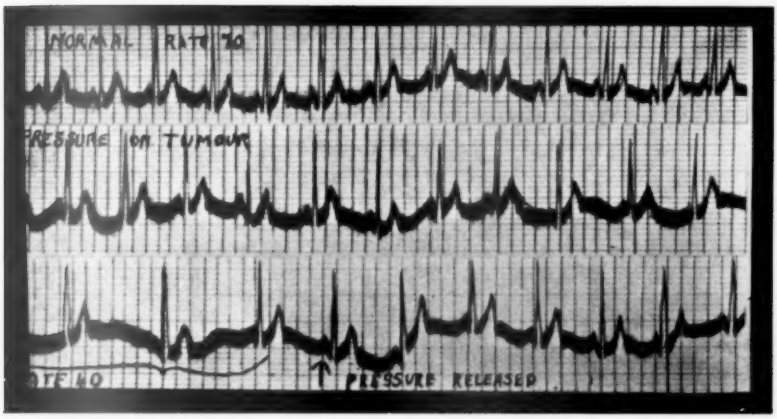

FIG. 2.—Electrocardiogram of Lead 1; pressure applied to tumour at beginning of second strip slows ventricular rate to 40 and stops auricle.

FIG. 3.—Tumour after excision (*diagrammatic*).

FIG. 4.—Tumour in section.

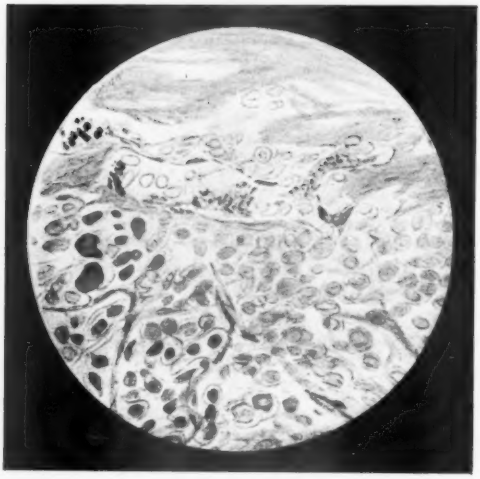

FIG. 5.—Drawing under one-sixth objective: shows infiltration of capsule and alveolar arrangement of large cells.

heart with syncope. The attacks were very sudden, and the patient believed they were brought on by movements of the head and neck.

Operation, February 20, 1928 (Mr. Page). Tumour found to envelop common, internal and external carotid arteries, the bifurcation being at about its middle. It was extremely vascular, and was excised together with the arterial trunks passing through it. In order to gain access to it the internal jugular vein had to be divided, and the cervical sympathetic was also severed. The vagus was clearly seen adherent to the back of the tumour, but was uninjured. Post-operative progress was uneventful, except for a transient aphonia and a persistent left enophthalmos and myosis. Patient has had no further syncopal attacks. Microscopical report on tumour: " hypernephroma type—must be regarded as malignant because of the type of cells, invasion of the capsule and growth in the lymph vessels " [1] (Professor L. S. Dudgeon).

Dr. Cassidy said that Mr. Page and he had looked into the literature of carotid body tumours, of which about one hundred cases were recorded up to the end of 1927. In the following three of these cases a carotid body tumour was associated with syncopal attacks: (1) *Knighton's case* (*New Orleans Med. and Surg. Journ.*, 1924, lxxvii, 237); (2) *Cade's case* (*Westminster Hospital Reports*, 1924, xix, 235-243); (3) *Boot's case* (*Ann. Otol. Rhinol.*, 1923-24, xxxii, 1241-5).

The exhibitors felt that operations for carotid body tumours should not be lightly undertaken, as the mortality of the operation seemed to vary between 25 per cent. and 30 per cent., and some of the patients who recovered were left hemiplegic or with paralysis of the larynx or tongue. They had asked themselves what were the indications for operation; these tumours were generally symptomless and of slow growth for many years, but later on might undergo malignant change, though the degree of malignancy was not great and metastases were rare and, if present, local. Resection in a young subject with a short history might be contemplated, for there was a possibility of removing the tumour without ligation of the carotids, and even if ligation were necessary, the chances of hemiplegia or death were not so great as they were in patients over the age of 30, in whose case operation was not justifiable in the absence of definite symptoms or evidence of rapid growth.

Mr. C. E. SHATTOCK said that at the Surgical Section recently he had shown a carotid body tumour which he had removed, and two other specimens were shown at the same meeting. The patients in all these cases had recovered.

Two Cases of Pulmonary Tuberculosis treated by Thoracoplasty.

By J. F. HALLS DALLY, M.D.

F. L. W., MARRIED WOMAN, aged 38, nullipara.

Family History.—Father died from pulmonary tuberculosis in 1920.

Past History.—1920, during sleep, slight hæmoptysis, repeated eight months later; no cough nor signs detected by private doctor; August, 1921, slight cough and loss of weight; December, " influenza and bronchitis "; April, 1922, hæmoptysis, 15 oz. during two days, T. B. +; May 11, 1922, admitted to Mount Vernon Hospital, emaciated, dyspnœic, weight 7 st. 8 lb. 12 oz. *Physical signs* of considerable infiltration, especially of apex and base of right lung, and in much lesser degree of left lung. August 8, X-rays showed adhesion to right dome of diaphragm with cavity in middle

[1] In respect of their size and the reticulation of their cytoplasm the tumour cells resembled those of the adrenal cortex rather than the cells of the adrenal medulla. No chromaffin tissue could be demonstrated in the tumour.

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of upper third of right lung surrounded by consolidation, and extensive infiltration of right base; October 26, discharged much improved, with right base clear of adventitious sounds. From May to October, 1922, temperature ranged from 97° F. in morning to 99·4° F. in evening, sputum 3j daily, T. B. +.

Attended as out-patient, and re-admitted to hospital March 2, 1926, by which time the right apex had become fibroid with a large cavity below clavicle, and infiltration of inner portion of left apex; July 1, 1926, phrenicotomy by Mr. C. E. Shattock (avulsion could not be performed owing to position of the nerve), after which the sputum became less, the temperature more settled and moist sounds fewer. Discharged August 14, 1926, having gained 9 lb.; sputum, 3ij T. B. +. Subsequently began to go downhill with loss of weight, T. B. + +, and hæmoptysis of about 6 oz. in July, 1927. Again admitted to hospital in October, 1927, and thoracoplasty decided upon. The first stage of this operation was performed by Mr. Shattock on October 27. Ribs 5 to 11, inclusive, were resected to a total extent of 24 in. on right side, with little pain and no vomiting, but some general reaction. Afterwards the condition remained good, sputum decreased, and signs in left lung became less active. The second stage was done on November 24, 1 in. of the first rib being removed and 1½ to 2½ in. of ribs 2 to 4 inclusive. Sputum increased temporarily, but there was less reaction than after the first stage. Subsequently, cough and sputum both decreased, and signs of excavation became less apparent. The pre-operation temperature ranged between 98° F. and 100° F., increasing after the first stage for twelve days, and then falling to the former level. For a fortnight after the second stage the temperature averaged 97·8° F. to 99° F. One month later it had fallen to within normal limits, at which it has remained. March, 1928, weight 8 st. 0 lb. 12 oz., general condition good, eats and sleeps well, weight steadily increasing, sputum steadily diminishing, on moderate exercise.

H. P., schoolboy, aged 16.

Family History.—Mother and one brother died from pulmonary tuberculosis.

Past History.—July, 1925, school doctor found signs of pulmonary tuberculosis and patient was admitted to Brompton Hospital for twenty-one weeks. On admission to Mount Vernon Hospital in February, 1926, cough and sputum were present with T.B. +; weight 4 st. 6 lb. 8 oz.; involvement of upper two-thirds of the left lung with signs of excavation to third rib and pleural thickening, also involvement of inner third of right lung from apex to root. Temperature remained unstable and sputum increased to 3ij on slight exercise, with no satisfactory gain in weight. Ten months later the heart and aorta had become displaced to the left, the right side clinically becoming dry, the temperature more stable, the weight constant at 5 lb. and sputum less but T.B. still +. Twelve lipiodol injections were given at weekly intervals with only slight benefit. By October, 1927, the area of excavation had increased so as to involve the outer third of the upper right lobe, the right side remaining dry. Left thoracoplasty was thought advisable, and was performed by Mr. Shattock in two stages, one month intervening between each. Ribs 4 to 11, inclusive, were first resected, 17½ in. in all, followed later by 1½ in. taken from ribs 2 and 3, and ¾ in. from the first rib. The condition after the first operation was excellent, the temperature returning to normal within a week with complete disappearance of sputum. After ten days the pulse-rate, which, before the operation, had for some time averaged 102 per minute, fell to a level of 68 per minute. Following the second stage there was more general reaction, but the temperature which was 100° F. for two consecutive nights fell in five days to normal, and from that time to the present there has been no sputum.

Present Physical Signs.—Left lung: dry fibroid creaking sounds. Right lung: few similar sounds posteriorly, probably conducted from left side. General condition

remains good, patient eats and sleeps well, is afebrile and without sputum or pains. To correct the lateral scoliosis which is likely to occur as a result of growth, an appliance has been fitted to hold the head erect and so to keep the spine in as straight a position as possible.

Remarks.—In both the above cases thoracoplasty was decided upon since: (*a*) a fatal result was considered to be inevitable within a comparatively short time unless the rapid march of the disease could be checked; (*b*) the condition of the opposite lung presented no contra-indication in the shape of extensive disease, and (*c*) the general condition was not already one of advanced toxæmia. Ultimate success or otherwise largely depends upon whether the infective process on the less affected side can be brought to a standstill or not, by increase in general and local resistance induced by checking absorption of toxic products from the gravely diseased opposite lung as the result of thoracoplasty.

Discussion.—Sir HERBERT WATERHOUSE (President) asked whether Mr. Shattock knew of a case in which the operation had been done on both sides, not, of course, at the same time. If bilateral tuberculosis existed, was there any reason why the operation should not be done on one side, and later on the other? Scoliosis resulted in adult patients as well as in children.

Mr. C. E. SHATTOCK said that the operation which he performed had followed the usual lines. He had tried to avulse the nerve in the case of the woman, but it had broken. With regard to the boy, the second stage was difficult owing to the approximation of the first three ribs which followed the extensive collapse caused by the first stage in a young patient. The boy was wearing a supporting jacket to prevent the development of severe scoliosis. Phrenic avulsion was done under local anæsthesia, through a transverse incision just above the clavicle. The skin incision was marked out before the patient's head was extended and turned to the opposite side. The left nerve was not so easy to find as the right, owing to its position and the proximity of the thoracic duct. Thoracoplasty was performed under gas and oxygen with a little ether. If more than a month elapsed between the stages, the divided ribs became fused and collapse did not proceed so fully after the second stage.

In answer to the President: he did not think it possible to do the operation on both sides in the same patient. The operation produced extreme collapse of the lung, which could be verified post mortem in cases in which the patients died some time after thoracoplasty.

Dr. HALLS DALLY (in reply) said he did not much like the operation to be performed on children, because of possible resulting deformity of the spine, but in this case the deformity was not great. The paravertebral route combined maximum compression with minimum disturbance.

Case of Total Transposition of Viscera.

By J. F. HALLS DALLY, M.D.

A. J. E., AGED 25, labourer, admitted to Mount Vernon Hospital as a case of quiescent tuberculosis involving right upper and lowest lobes, and left apex. Sputum negative on nine successive examinations. Clinically he was thought to have total transposition of viscera which (so far as radiological evidence can go), was confirmed by the radiograms exhibited. Radiograms of a similar condition in a woman under the care of my colleague, Dr. Basil Price are shown for comparison.

Remarks.—Since the introduction of X-ray investigations, the condition appears far less infrequently than was previously believed. Recent figures state the relative frequency of discovery of transposition as follows: X-ray investigation, 1 in 1,400 cases examined; Post-mortem, 1 in 5,000; Dissecting room, 1 in 10,000; Physical examination, 1 in 35,000. The developmental causes are unknown. It is probable that total transposition is not limited to the thoracic and abdominal viscera, kidneys and adrenal glands, but may include all somatic structures. Health is not impaired

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from the point of view of life assurance or strenuous occupations. Additional diagnostic evidence is afforded by electro-cardiograms, which show inversion of ventricular complexes indicating right-sided preponderance, which may also, however, be induced by dextrocardia alone, due to (*a*) transposition, (*b*) retraction, with X-ray signs of right-sided fibrosis, (*c*) pressure to right by fluid (left pleural effusion), air (left pneumothorax) or solid (new growth on left side of chest).

Two Cases of Familial Acholuric Jaundice in Brothers.

By Adolphe Abrahams, M.D.

I. S., aged 11½, H. S., aged 10, brothers.

At eighteen months, colour of urine in the case of I. S. noted to be dark brown with gradual return to normal. Since then similar attacks at irregular intervals; one two years ago followed measles, another one year ago after influenza. The average duration of each attack is four days. The attacks are preceded by irritability, restlessness, sleeplessness, and gradually increasing pallor and weakness develop. It is doubtful what degree if any, of pyrexia is present.

H. S. was jaundiced two days after birth. At the age of 3, dark urine was noted. Since then he has had attacks at irregular intervals similar to those of his brother. The attacks appear to have been more frequent in cold and changeable weather, and the mother was convinced that they were synchronous in the two cases. This phenomenon does not bear close investigation, and certainly during a severe attack in the case of the younger boy (H. S.) in February last, I. S. was unaffected.

There is no history of similar symptoms in the relations on either side. An elder brother, aged 13, has never suffered, he has not an enlarged spleen and his blood-count is substantially normal.

Both boys are sallow and with an icteric tinge. The tonsils are large in the case of I. S. The spleen is enlarged, more so in the case of H. S., otherwise no abnormality is discoverable. Blood-pressure, I. S., $\frac{110}{85}$, H. S., $\frac{125}{90}$.

The urine in both is free from albumin, sugar, bile-salts and pigments.

Blood Examinations.—The Wassermann reaction in both cases is negative.

Van den Bergh Test.—In I. S., bilirubin 5 units, negative direct and delayed positive indirect reaction. In H. S., bilirubin 2·5 units, negative direct and delayed positive indirect reaction.

Hæmoglobin, I. S., 77·5 per cent.	H. S., 85 per cent.
Erythrocytes, I. S., 4,500, 000.	H. S., 4,780,000 per c.mm.
Colour index, I. S., 0·84.	H. S., 0·9.
Leucocytes, I. S., 7,800.	H. S., 4,700 per c.mm.
Polymorphs, 77 per cent.	Polymorphs, 71 per cent.
Lymphocytes, 22 per cent.	Lymphocytes, 27 per cent.
Monocytes, 1 per cent.	Monocytes, 2 per cent.
Platelets, I. S., 320,000.	H. S., 400,000 per c.mm.

Fragility test.—In both cases hæmolysis occurs in 0·4 per cent. saline, not in 0·45 per cent. The coagulation and bleeding times were not investigated.

Despite the absence of increased fragility of the red corpuscles a diagnosis of familial acholuric jaundice seems the most likely. Is splenectomy advised at the present time? There can be no question that very severe hæmolysis occurs during the attacks.

Discussion.—Dr. F. PARKES WEBER said that in familial groups of hæmolytic (acholuric) jaundice (such as the large one investigated by Gänsslen, of Tübingen) abnormal fragility of the erythrocytes was not invariably present, that was to say, in some (atypical) cases that sign was absent, as in Dr. Abrahams's present cases. Nevertheless, before deciding on splenectomy, it would be well to confirm the diagnosis by examining the two brothers during one of their attacks (instead of between the attacks) in regard to erythrocyte-fragility and other signs.

Dr. BERNARD MYERS said that neither of the two cases was typical, and he advocated waiting a little longer for further investigation. Concerning splenectomy, it would appear not to place the body at a disadvantage, even if the diagnosis were not sustained. On the other hand, waiting for another attack would probably be safe, as he had lately had in hospital a girl of 11 years, with typical acholuric jaundice of five years' standing, who had a temperature of 103° F., a red-cell count under 2,000,000, and *in extremis* on admission, and who yet made a perfect recovery after blood-transfusion followed by splenectomy.

Dr. DONALD HUNTER suggested that the reticulated red corpuscles might be estimated. In many cases of familial hæmolytic icterus the proportion was high, even as high as 30 per cent. This, of course, was not a pathognomonic sign.

In a series of thirty-four cases, Lord Dawson had analysed the results of treatment, and they were such as to favour surgical intervention very strongly. Of fifteen cases treated by splenectomy fourteen were alive and well, some of them many years after operation. One had died as the immediate effect of a surgical mishap. Of the nineteen not operated upon nine had died either of a severe hæmolytic anæmia, or of the complications of gall-stones. This liability to severe anæmia was a great danger, and might occur at any age. In one case of the series the patient suddenly became very anæmic, and three transfusions of blood did not save her.

Dr. ADOLPHE ABRAHAMS (in reply) said he intended to advocate operation in these cases. He had seen the younger boy three months ago, and the anæmia was of such alarming degree that he thought recovery could not occur without transfusion. But the mother was more optimistic in view of previous attacks that she had witnessed, and her judgment and prognosis were superior to his own. Nevertheless he could not reconcile himself to the prospect of many such hæmorrhages.

Case of Hypothyroid Heart.

By B. T. PARSONS-SMITH, M.D.

F. H. G., MALE, aged 56.

History.—Attended hospital February last, complaining of fainting attacks (usually associated with palpitation) and progressively increasing physical weakness; had been putting on weight during the previous year; memory deteriorating and nervous control impaired; hair recently brittle; cold, dry skin; speech rather slower than formerly and voice somewhat raised.

Examination.—Heart slightly enlarged; feeble apex impulse in the fifth space 4 in. from the mid-line; regular rhythm; sounds toneless at the apex; second sound relatively accentuated at the aortic region; pulse 64; blood-pressure 186 mm. systolic, 100 mm. diastolic; electro-cardiogram (fig. 1) ventricular complexes of low voltage; T-waves flattened in lead I and inverted in leads II and III; urine analysis negative.

Progress.—Considerable improvement under thyroid medication to date; effort syndrome definitely relieved; steady reduction in the weight (roughly 3 lb. per month); no return of the fainting attacks; myocardial efficiency more satisfactory, the electro-cardiogram (fig. 2), of April 11, showing clearly defined positive T-waves in all the leads.

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The clinical record and the electrocardiographic findings in this case suggest that the hypotonic state of the general musculature in hypothyroidism may be likely to involve the myocardium also.

Improvement in this case was associated with the appearance of well-developed T-waves in the cardiographic curves, which incidentally suggests that flattening of

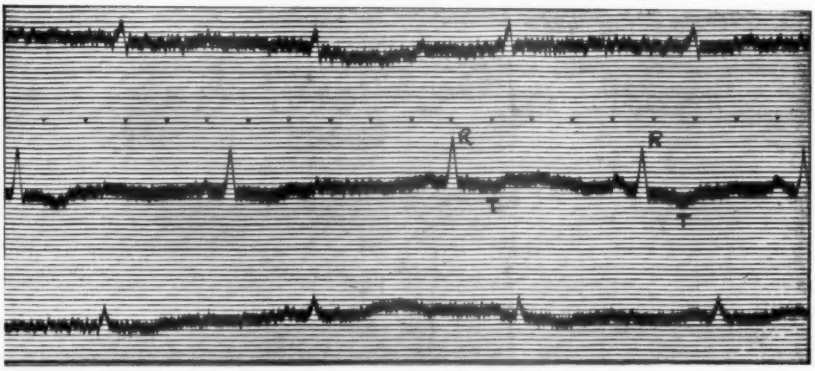

FIG. 1.—Ventricular complexes of the low-voltage type; T-waves inverted in lead II, flattened in leads I and III.

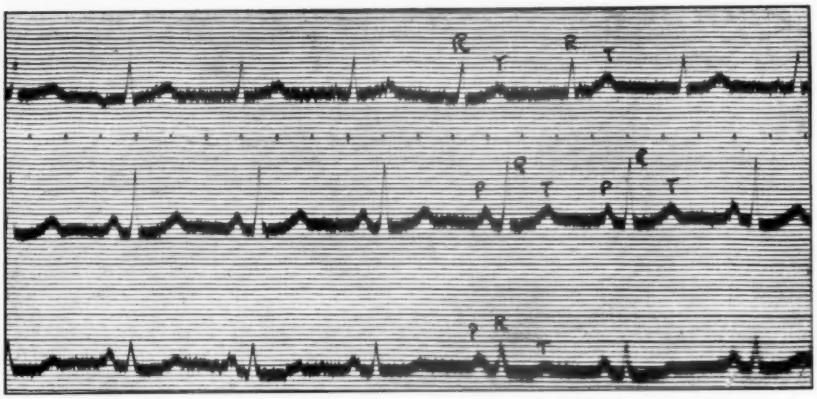

FIG. 2.—Curves recorded after treatment with thyroid extract; well developed T-waves in all the leads.

T-waves is not necessarily indicative of permanent pathological changes in the heart muscle, but that, under certain circumstances, it may be the outcome (possibly temporary) of diminished tone in the myocardium.

Discussion.—Dr. F. PARKES WEBER asked Dr. Parsons-Smith whether the thyroid treatment had lowered the blood-pressure in the present case, and whether it usually did

so in similar cases of hypothyroidism with relatively high blood-pressure. He believed that a considerable proportion of old myxœdema patients showed high blood-pressure.

Dr. PARSONS-SMITH (in reply) said he agreed with Dr. Parkes Weber. 186 systolic was not typical of hypothyroidism; usually in the latter condition the blood-pressure was subnormal. And one would expect that with a recovery from the hypothyroid state there would be a rise in the blood-pressure. That, however, did not put the diagnosis out of court, because in true myocardial degeneration one often saw a raised blood-pressure, in fact, he believed that in the majority of cases of true myocardial degeneration there was a raised blood-pressure.

Case of Obesity.

By B. T. PARSONS-SMITH, M.D.

R. J., FEMALE, aged 40.

History.—Increasing tendency to physical weakness and exhaustion with effort during the past five years; breathlessness, palpitation and recurring attacks of giddiness; also complains of pain in the left side of the chest (not associated with effort) which radiates to the shoulders and generalized pains in the arms, left leg, etc., the latter at times involving definite transient hemiparesis (left).

Past History.—Perfectly healthy in infancy and early childhood; rheumatic fever and scarlet fever at nine years; married at 18 (weight at this time 8 st. 6 lb.); first child born at 19, normal pregnancy; began to gain weight following the pregnancy and has continued to date (present weight 14 st. 8 lb); periods regular but loss always scanty; appetite always large but recently excessive; definitely more thirsty recently.

Clinical Examination.—Heart not enlarged (confirmed by X-ray); rhythm regular except for occasional premature contractions; soft systolic murmur at the apex; sounds at base relatively muffled and toneless in type; pulse 88, occasional intermissions; blood-pressure 170 mm. systolic, 90 mm. diastolic; slight tendency to cyanosis of the lips but no gross venous congestion; no œdema.

Electro-cardiogram.—T-waves flattened in all the leads. Urine analysis: specific gravity 1028; sugar present, no albumin; no diacetic acid. Blood-sugar (specimen taken three hours after meal) = 0·13 per cent.

The present signs and the past history suggest that the condition may be one of endogenous obesity secondary to lack of endocrine balance, or, on the other hand, that it is a case of adiposis dolorosa (Dercum's disease.)

Discussion.—Dr. STOLKIND said he thought this was probably a case of Dercum's disease. A skiagram of the sella turcica region should be taken to see whether there was any enlargement. He advocated multiglandular treatment.

Sir HERBERT WATERHOUSE (President) said he also thought it was Dercum's disease.

A Large Exostosis of the Humerus in a Girl aged 10 years.

By CECIL P. G. WAKELEY, F.R.C.S.

ROSE B., aged 10, came under observation in March, 1928. Her mother stated that she had a fall in December, 1927, and after that always complained of pains in the right arm. In February, 1928, she noticed a lump in the upper part of the right arm, and it was because of this the child was brought up to hospital.

On examination there is a large hard swelling attached to the upper third of the right humerus. It projects backwards and outwards. It is not painful on pressure, although the patient states that it has been so in the past.

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X-ray examination reveals an exostosis, with a very large pedicle.

The points of interest in this case are:

(1) The history of injury three months before the case came under observation. The exostosis could not have been formed in three months, as it was quite two inches from the epiphyseal line.

(2) The large size of the exostosis and its distance from the epiphysis.

Sir HERBERT WATERHOUSE (President) said this case upset the idea of the cell-rest. He had never seen such a tumour so far from the epiphyseal line, nor did he remember an exostosis of this nature with so large a base (2 in.). He would like to see a report of the case after the exostosis had been removed.

Section of Dermatology.

President—Dr. J. M. H. MacLeod.

[*March* 15, 1928]

CASES.

Exfoliative Dermatitis with General Pigmentation.

By W. J. O'Donovan, M.D.

H. R., aged 61, general labourer, married; wife alive and well; three children, two well, a third has chronic rheumatism.

Family History.—No miscarriage or skin disease.

Patient is abstemious: denies venereal disease. His skin was quite clear until the age of 25, when an attack of redness and scaling affected his face, ears and feet. After a month's in-patient treatment in Reading Hospital he returned to work and remained well except for rare attacks of influenza with abdominal pains and diarrhœa.

In October, 1926, when he was employed in a timber shop, carrying teak, deal, and mahogany, and clearing up sawdust, an eruption began on the extensor surface of the forearms. At first the lesions were small pimples; they were dry and itched greatly. Later, the legs were affected below the knees, then above; in spring, 1927, his face was affected. Finally the trunk became affected, in October, 1927, and he says he has become steadily worse since then. He thinks he has lost over a stone in weight and has been thirsty throughout.

He first attended the London Hospital, October 11, 1927, showing a purplish-brown pigmentation of the trunk and limbs with white islands of natural colour, generalized exfoliation with fissured palms, wizened features and slight dropping of both lower lids. There were numerous deep scratch marks everywhere.

February 2, 1928 (*in Hospital*).—He showed no obvious wasting. The hair of the outer part of the eyebrows had dropped out, also most of the eyelashes. The face was smooth and expressionless, with slight ectropion of both lower lids. His face and scalp were bright pink, and the skin everywhere was dry and cracked, with adherent scales. The desquamation was in thick flakes, particularly over the scalp and ears. From the neck downwards on to the trunk there was a burnished silvery-brown darkening of the skin.

Lungs, heart, liver and spleen: no abnormalities; patient always hungry.

Upper and lower limbs stiff but knee-jerks present. Pupils: equal and active. Tongue: clean and moist. Teeth: a few carious stumps. Chest: kyphosis. No adenopathy; no pyrexia; urine normal. Wassermann reaction negative. Blood-pressure, $\frac{143}{80}$ mm. Hg.

Fractional test-meal showed no free hydrochloric acid—total acidity 10-16 over $2\frac{1}{2}$ hours.

Direct and indirect van den Bergh's reactions both negative. Fæces: no parasites seen.

Blood-counts.—(1) January 3, 1928: erythrocytes, 4,800,000; hæmoglobin, 70 per cent.; colour index, 0·73; leucocytes, 13,200; [*Differential*] polynuclear neutrophils, 84·5 per cent.; polynuclear eosinophils, 4·5 per cent.; small lymphocytes, 6·5 per cent.; large lymphocytes. 2 per cent.; large hyaline cells, 2 per cent.; coarsely granular basophilic cells, 0·5 per cent.

(2) March 6, 1928.—Erythrocytes, 3,500,000; hæmoglobin, 55 per cent.; colour index, 0·77; leucocytes, 11,000; [*Differential*] polynuclear neutrophils, 82·5 per cent.; polynuclear eosinophils, 2·5 per cent.; small lymphocytes, 10·5 per cent.; large lymphocytes, 0·5 per cent.; large hyaline cells, 4 per cent.

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Report on Histological Examination.—Chronic inflammation of skin of pectoral region; elongation and broadening of epidermal papillæ; stratum corneum nucleated throughout with ordinary spindle nuclei, also containing unusual large, round, granular masses of chromatin. Exfoliation of nucleated strips of stratum corneum; no abnormality in stratum granulosum. Much focal degeneration with pyknosis and quite abundant mitosis in stratum Malpighii. Moderately richly cellular granulation tissue in papillary and subpapillary zones or dermis compound of numerous spindle, round and oval and multinucleate fibroblasts or "histiocytes," many of them packed with melanin pigment, numerous dilated capillaries and few lymphocytes and neutrophil leucocytes.

Progress.—Pigmentation has become much deeper and has extended over the face and down the limbs since admission to hospital. The pruritus has remained constant and distressing; desquamation, which was extensive (handfuls), is now much less.

Dr. May, of the Royal Berkshire Hospital, Reading, has informed me that this patient was treated with arsenic for a varying period in 1926 and 1927, the maximum dose never exceeding three minims, given thrice daily; the total period of administration was about six months. Silver salts were not given; pigmentation was first noted on October 4, 1927.

I think it is sufficient to record this case of exfoliative dermatitis in which grave general pigmentation has developed and which shows evidence at present of severe anæmia, but in which neither leukæmic changes nor those of pernicious anæmia have yet appeared.

Discussion.—Dr. J. M. H. MacLeod (President) said he concluded that the pigment was melanin. [Dr. O'Donovan: "Yes."] He did not remember to have seen exfoliative dermatitis followed by such pigmentation. He thought the skin was altered as well as pigmented; it was certainly withered.

Dr. S. E. Dore said he had never previously seen exfoliative dermatitis with pigmentation.

Dr. W. N. Goldsmith said that in a case in University College Hospital, under Dr. Gray, there had been a similar appearance. In that patient the pigmentation was slaty-blue, and in parts de-pigmentation could be seen; there were large areas of very white skin. It had begun as exfoliative dermatitis. There was no history of arsenic; the patient had leucocytosis when admitted, but while in hospital it became less. He had had peculiar attacks of fever which ended when he had hexamine, hence the fever was thought to be connected with his cystitis.

Dr. F. Parkes Weber asked whether there had been vomiting or fainting, or whether signs of general cachexia were developing. If, in spite of recovery from the exfoliative dermatitis, this patient were going downhill, there might be true Addison's disease present, or else some disturbance of the solar plexus causing the pigmentation.

Dr. W. Jenkins Oliver asked what treatment this patient had received before going to London Hospital; did it include chrysarobin or tar? [Dr. O'Donovan: Boracic ointment is all I know of.]

Dr. Semon said he thought that a patch of pigment on the left buccal mucous membrane in this patient was a point against the arsenical ætiology.

Dr. Alva Benjamin said he had had a case of exfoliative dermatitis in an Australian patient. When he saw her, last October, her temperature was 101-103° F., the exfoliative dermatitis was universal, very red and with much scaling; irritation was severe and she had cold sweats. Less than a week after treatment with small doses of arsenic the temperature became normal—the doses were $\frac{1}{500}$ gr. thrice daily—but the skin did not clear very much. No organism was found in the urine; the pathologist said it was unnecessary to take a blood-culture, as if there were an organism in the blood it would appear in the urine too. Six weeks after the temperature had become normal a direct blood-culture was done, and produced a pure culture of *Staphylococcus aureus*. A special vaccine was prepared and was given by the mouth. She had one dose only at the end of last December, and since then had gone quickly ahead; her skin, except in isolated spots, was now as smooth as before she had the condition and she felt quite well generally. He did not know whether it was usual to find an organism in the blood in such cases.

Dr. GRAHAM LITTLE said he agreed that the pigmentation might be arsenical. He had seen very extensive pigmentation after only three-minim doses of arsenic given thrice daily for two months in a case of pemphigus, and he had been compelled to abandon the medication.

Granuloma Fungoides. ? Case for Diagnosis.

By H. C. SEMON, M.D.

PATIENT, aged 61, Commander R.N. (ret.).

History.—Has always had a dry skin. When aged 15, and still on the *Britannia*, first noticed a dry scaly eruption on lower abdomen, flexor aspects of elbows and knees, and slightly on back. Is certain there was no itching either then or subsequently. Various practitioners, including the late Dr. Liveing, diagnosed psoriasis. The condition had never entirely cleared up, but persisted as "blotches and spots" until early in 1927, when the character of the eruption seemed to change. It became more red and angry-looking, especially on the legs and the back of the thighs, and appeared to be raised above the surface. Other parts of the body were attacked, and a patch over the precordium, alleged to have been rubbed by carrying a golf-bag under the arm, "became raw and began to discharge."

The strap of the bag is alleged to have caused similar ulceration on left shoulder. Twelve or more spots have broken down similarly and healed extremely slowly under a scab.

Two types of lesion are present: (*a*) Reddish-fawn coloured, flat, slightly scaly patches, of various sizes, shapes and degrees of soft infiltration. Some resemble a well recognized type of seborrhœic dermatitis.

(*b*) Flat, raised, soft nodular lesions, varying in size from pea to thumb-nail with pronounced tendency to necrose, and heal exceedingly slowly under a relatively adherent scab, from the margins of which small quantities of sticky mucoid pus are occasionally discharged.

The mucous membranes are unaffected, and there is no recognizable enlargement of glands.

The Wassermann reaction is negative. A blood-count shows no variation from the normal.

Examination of smears of the discharge and cultural tests have revealed a hæmolytic staphylococcus from which a vaccine has been prepared.

The usual response to X-ray treatment of the ulcers and infiltrations is present in a minor degree only, and is delayed.

Report on biopsy of a small early unbroken nodule (Dr. Embleton):—

"Each of the pieces of tissue on section shows appearance of a chronic inflammatory change. The subcutaneous tissue shows marked fibrosis and is infiltrated with leucocytes and plasma cells. The epidermis shows some simple hyperplasia of a warty type. No evidence of any malignant change."

The points against the conditions being mycosis fungoides are: (1) The absence of itching at any period of the case (I believe, however, that cases of mycosis without itching have been reported); (2) the absence of, or delayed response, of the necrosing nodules to X-rays; and the fact that the nodules are not of the tomato-like, hyperplastic form usual in that disease.

The histology neither supports nor excludes the diagnosis, and therefore affords no help. I confess inability to offer any diagnosis other than that above suggested.

Dr. J. M. H. MACLEOD (President) said there was not much evidence for a diagnosis of mycosis fungoides. Several lesions seemed to have disappeared under treatment, or spontaneously; this was very rare in mycosis fungoides. Further, the microscopical

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appearances did not suggest that condition. In the epidermis there was scaliness, with abscess formation in the central layers; this did not occur in mycosis fungoides, and the corium changes were not sufficiently definite to warrant conclusions. He was not sure that the condition was not pustular psoriasis.

Unilateral Erythro-cyanosis Crurum Puellarum.

By F. Parkes Weber, M.D.

The case is one of "erythro-cyanosis (frigida) crurum puellarum," according to prevailing Continental nomenclature,[1] limited to the right leg, which is presumably a locus minoris resistentiæ, owing to infantile paralysis in early life.

The patient, E. K., now aged 20, first came to my attention in May, 1927, on account of the chronic erythematous condition of the lower part of the leg, which I had discussed in 1925.[2] The condition in this case was limited to the right leg, which was slightly smaller in circumference and length than the left, probably as a result of infantile paralysis in early life.

The erythema or erythro-cyanosis had been present for the last two and a half years, but had been less noticeable in summer. She was "plump" and apparently free from other complaints. In particular there were no signs of anything wrong in the thoracic organs or in the blood-count; the brachial blood-pressure was, systolic, 130 mm. Hg; diastolic, 95 mm. Hg. The blood-serum gave negative Wassermann and Meinicke reactions, and the Pirquet cuti-reaction for tuberculosis was negative. The local condition in the leg was much improved by a period of rest in bed.

The erythro-cyanosis is, however, still present in the lower part of the right leg, especially purplish swellings in the ankles behind the malleoli on both sides of the tendo Achillis. Moreover, from sitting at the fire, she has now developed a marked *erythema reticulatum ab igne* in both legs; this is of only about three months' duration.

I think that the easy development of a high degree of erythema reticulatum suggests that there is a constitutional defect in the peripheral circulation (veins and capillaries) in the present case, and I think that the unilateralism of the erythro-cyanosis points to the constitutional defect being greater in the right leg, probably as a result of previous infantile paralysis in that leg. In regard to the occasional supervention of true (tuberculous) erythema induratum (Bazin's disease) in erythro-cyanosis cases it is interesting that in the present patient the Pirquet cuti-reaction for tuberculosis was negative.

I have lately had reason to believe that an erythromelalgic condition of a hand, with paroxysms of intense flushing and local heat, may supervene on a previous chronic chilblainy condition; and it is possible that in severe chronic erythro-cyanosis cases similar erythromelalgia-like flushing may supervene, owing to involvement of the peri-arterial sympathetic nerve supply. I now regard erythromelalgia[3] as a syndrome due to a chronic inflammatory state of the peri-arterial sympathetic nerve supply to the affected part. The operation of peri-arterial sympathectomy (or some modification) has been tried in at least two erythro-cyanosis cases, with no beneficial results, but it does not follow that it might not be of service in some erythromelalgic cases, in which the peri-arterial sympathetic

1 Cf. A. V. Karwowski, *Dermat. Wochenschr.*, Leipzig, 1927, lxxxv, p. 1161, and A. Alexander, *ibid.*, 1927, lxxxiv, p. 601.

2 F. Parkes Weber, *Brit. Journ. Dermat. and Syph.*, London, 1925, xxxvii, p. 259; and *Proc. Roy. Soc. Med.*, Section of Dermatology, 1926-1927, xx, p. 61; also *Proc. Roy. Soc. Med.*, Clinical Section, 1928, xxi, 84.

3 F. Parkes Weber, "Erythromelalgia," *Med. Press*, London, 1927, clxxv, p. 266.

nerve supply may be diseased, the attacks of heat and flushing being due to paroxysmal exacerbation of the disease.

Discussion.—Dr. A. C. ROXBURGH, referring to Dr. Weber's remarks on the von Pirquet test, said that since last autumn he had been collecting these cases and carrying out the test on all cases of erythro-cyanosis and ordinary Bazin's disease which were seen. At one time he thought he would find that the Bazin cases gave a positive, and the erythro-cyanosis a negative von Pirquet reaction. But a larger series showed that was not the case. In some cases it was difficult to decide in which group to place the condition. Three patients came to hospital on two successive days, and neither Dr. Adamson nor himself knew certainly into which group to put them, whether the condition was of the nodular Bazin or of the diffuse type, as it had to some extent the characters of both.

Dr. H. SEMON said that in surgery peri-arterial sympathectomy was performed for gangrene and conditions like Raynaud's disease, a spasm of smaller arteries, and he thought that it would aggravate the condition in Dr. Parkes Weber's case. There was probably in these cases a paresis and dilatation, not a constriction, and intramuscular injections of pituitary giand might be tried. In one of his cases there was definite improvement, especially subjectively.

Dr. A. M. H. GRAY said he had discussed this question with Sir Thomas Lewis in connexion with an apparently genuine case of erythromelalgia, which was getting worse. The patient's doctor had written to him asking if the speaker thought sympathectomy would be of value. Sir Thomas gave, on theoretical grounds, the opinion just expressed by Dr. Semon, that the operation would have the opposite effect, as it would increase the arterial blood flow.

Chronic Superficial Glossitis with Black "Fur."

By W. N. GOLDSMITH, M.D.

PATIENT, a man aged 60, in October, 1925, had epileptiform fits, during one of which he bit his tongue which then became sore. Gradually a thick fur developed and became darker and darker. The tongue has remained sore and covered with black "fur" ever since.

All the patient's teeth have been extracted; they were very bad when the trouble began. The Wassermann reaction is negative. There is osteo-arthritis of the lumbar spine.

Bacterial examination of scrapings of tongue (November, 1927,) gave cultures of *Nocardia lingualis* and an organism of the saccharomyces variety, *Cryptococcus linguæ pilosæ* (see below). No other organisms developed on culture.

I first saw the patient in December, 1927. Almost the whole of the tongue was heavily covered with black fur. Frequent massaging with an ointment containing monsol soon cleared the anterior part. But at the back the condition persists. He still complains of great soreness on the dorsum near the tip, where there is now no fur. A skiagram showed excessive peribronchial fibrosis over both lungs, but no mottling; no evidence of tuberculosis; old empyema scar at right base.

Injections of neosalvarsan have had no effect. The patient is under-weight, but is now steadily gaining, though he cannot manage a normal diet. In a bacterial examination one month ago growths were obtained identical with the previous ones.

Black hairy tongue is not generally accompanied by marked subjective symptoms or other obvious change in the tongue. It is here associated with chronic glossitis. Recently I saw another case of black hairy tongue, in which the tongue was otherwise normal. In that case no unusual organisms were cultured, and it cleared up very rapidly under massage with monsol ointment.

Bacteriology.—The cultures were obtained for me by Dr. Ronald Ralph. They consist of two organisms identified as (1) *Cryptococcus linguæ pilosæ* (Lucet, 1901)

(= *Saccharomyces linguæ pilosæ* Lucet, 1901). First found by Raynaud, first studied by Lucet. Double-contoured blastomycetic bodies, 3 to 6 microns in diameter, are found on the surface and in the thickness of the hypertrophied papillæ. Grows best on a glucose or lævulose acid medium between 25° and 35° C. It ferments sugars and produces acid. In cultures the cellules may become elongated and suggest mycelium. It is generally found (*always*, according to Ralph) associated with *Nocardia lingualis* (= *Actinomyces guegueni*).

(2) *Actinomyces guegueni* (Brumpt). Described in 1908, by Guéguen, as *Discomyces lingualis*. In the hypertrophied papillæ of the tongue the mycelial filaments are fragmented and resemble bacilli. On culture one may obtain chains of conidia of oöspora type.

Ralph thinks that these two organisms represent a true symbiosis. He has found them together in three other cases of black hairy tongue, and has not met with them in any other condition. In three of the four cases there was a definite history of injury, e.g., during tooth extraction. An acid mouth would favour the infection. These organisms have never been proved to be the cause, but there is much to suggest that at least one type of black hairy tongue is attributable to them. As opposed to *Rhizopus niger*, these organisms themselves produce no pigment.

Dr. J. M. H. MACLEOD (President) said there seemed to be several types of black, hairy tongue, but they were probably all of fungus origin, and not simply related to the so-called "geographical tongue," in which white patches came and went spontaneously, associated with an irritable gastric mucosa.

The question of the organisms found in the present case was difficult as the *Cryptococcus linguæ* was a fairly prevalent organism, and he was not sure that it was pathogenic. The nocardia was a different matter, and seemed a more likely cause. He suggested painting the patches with 1 per cent. salicylic lotion.

? Pityriasis Rubra Pilaris. Case for Diagnosis.

By E. G. GRAHAM LITTLE, M.D.

THIS man has had this eruption of follicular papules with deep brown staining almost continuously since 1896. He was seen by Dr. Wyndham Cottle, who diagnosed lichen planus. The acneiform lesions on the upper shoulders are recent, and are not concerned with the chronic persistent eruption. Was the eruption lichen planus, and is the present disease pityriasis rubra pilaris? This seems more probable; the duration of the eruption is against the diagnosis of lichen planus.

Dr. S. E. DORE said that the condition might be pityriasis rubra pilaris, but there were neither the usual plugs on the phalanges nor scaling of face or scalp. He was told that this patient had been using sphagnol for years, but he did not think that had caused the eruption.

Lichen Planus.

By E. G. GRAHAM LITTLE, M.D.

THIS patient is a girl with a very curious eruption of large, almost nodular lesions, confined to both axillæ, with one or two typical lichen planus papules at the edge of the larger group of lesions; and with another extraordinary condition which one would call hypertrophic lichen planus if it were elsewhere than upon the forehead. I have never seen hypertrophic lichen planus on the forehead. There is nothing in the mucosæ or anywhere else on the body, except one doubtful patch on the middle of the chest, a round, rather atrophic lesion, which might be due to a scald or burn, but more probably is a lesion of lichen planus annularis.

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Hæmangiectatic Hypertrophy (Parkes Weber).

By A. M. H. GRAY, C.B.E., M.D.

THIS is a case of extensive congenital nævoid condition of the right lower limb in a boy aged 10½. The abnormality has been noted since he was two or three years old. I first saw him some months ago at Great Ormond Street Hospital, where he had seen a medical and a surgical colleague of mine (the surgeon because of the large pigmented patch on the front of his shin, which was becoming larger). In addition, he had a vascular capillary nævus behind the thigh, above the popliteal space, in the same limb. On inspection we saw that the right limb was larger and darker than the left; there were dilated veins in the region of the knee. At University College Hospital, Sir Thomas Lewis investigated the case thoroughly from the point of view of the condition of the circulation in the leg. The right lower limb is 4·5 cm. longer than the left, most of that increased length being in the leg, not in the thigh. The increased length of the femur is 1 cm., and below that 3·5 cm. There is also a difference in girth, that of the right limb being greater by 1 in. Pulsation in the right femoral artery is greater than in the left. On the right side this can be felt down to the popliteal space, on the left, only 2 in. below the groin. There is an area of abnormal pulsation internal to the patella, and extending down the anterior surface of the leg. The pulsation disappears on compression of the femoral artery. There is a thrill in the right femoral artery and over the enlarged vessels under the patella which, Sir Thomas Lewis thinks, is of arterial origin. There is also distinct pulsation in the veins. Over the femoral vessels there is a continuous murmur. The right ankle is of the same temperature as the left, but in the region of the right knee there is a difference of 2·7° C., and at the right thigh 2·1° C. The right leg is 3·75° C. hotter than the other. Obliteration of the right femoral artery has much greater effect on the pulse-rate than obliteration of the left femoral artery, indicating lesser resistance to outflow on the right side. There is a general increase in the circulation of that limb, and probably a communication exists between arteries and veins, though Sir Thomas Lewis is not certain how that comes about; he does not think there is any marked capillary dilatation.

One or two features are of clinical interest. With regard to the pigmentary patch on the front of the leg, a portion was recently examined. It now looks like a pure pigmentary patch, though it then seemed very vascular, like Kaposi's sarcoma, and I thought we should find a large increase of vessels, including capillaries. However, sections showed little increase of vessels, neither was there much pigmentation. All the extra pigment seemed confined to the basal layer of the epithelium; nævus cells were absent. This patch has been evident only two years, and I wondered if it was a commencing melanoma likely to become, or already, malignant.

Should this limb be treated as a whole, or should we devote ourselves to the pigmentary patch? The centre of the patch has shown warty thickening, and there has been surface damage, which has cleared up. There is still warty thickening and some crusting.

Suggestions for treating the limb as a whole are not, I think, very satisfactory. One suggestion is amputation, another is ligation of the main artery or of some branch of the femoral. The surgeons who have seen the case fear that if anything of that sort is done the circulation of the foot will be so much diminished that gangrene of the toes may ensue, and they do not think the condition bad enough for amputation. The original suggestion was to cut out the patch and to take a graft from another part of the body. The surgeons are not anxious for a cutting operation, as they doubt whether they could stop hæmorrhage.

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In view of the possibility of it being malignant, should this lesion be destroyed by diathermy, or by steam cautery? Or should one conclude that it is malignant and recommend amputation?

Note.—Further examination of the sections, together with the history of the late appearance of the pigmentary patch, suggests that it is not of nævoid, but of inflammatory origin. Some parts of the original pigmentary area are disappearing spontaneously, and a few new spots have developed around the old ones in obviously inflammatory papules.

Discussion.—Dr. F. PARKES WEBER said this case belonged to a class in which he had taken much interest for many years, and to which he had referred in various discussions. Finally, he had named it "hæmangiectatic hypertrophy" of limbs, and in the *British Journal of Children's Diseases*[1] he had collected all the cases he knew of bearing on the subject. These cases had the peculiarity that the developmentally larger limb often showed some form of vascular nævus. The most interesting feature here was the pigment in the nævus. He knew of no other case of hæmangiectatic hypertrophy with a definite pigment nævus. Some of these cases had attracted attention on account of congenital "varicose" veins. Others had had enlarged arteries, and some cases had been named "phleb-arteriectasis," because both vein and artery were enlarged. In some cases there was a thrill or pulsation to be felt in the veins, whether or not there was genuine anastomosis between arteries and veins. He had included all the cases of this kind in his series, and in his article he had pointed out that the condition of cirsoid aneurysm in early life might be related to congenital phleb-arteriectasis. He believed that in very bad cases of the condition operations had been performed. He did not think amputation desirable in the present case, unless it was considered that it would prevent the possible future development of malignant melanotic tumour, which was unlikely to develop.

In his paper on the subject he had separated "hypertrophy of the hemi-hypertrophy type" from cases that he included under "hæmangiectatic hypertrophy of limbs."

Dr. SYDNEY THOMSON said he had seen two similar cases, but without pigment. Only when the patient was stripped, in bed, and the condition was viewed from some distance, was it realized that there was a true hemihypertrophy. This was difficult to appreciate in the upper limb. In most cases the changes in the leg were unnoticed until the child was over 2 years old. It might be for static reasons that the hypertrophy then became more marked in the lower limb. In the cases he had seen there was true increase of bony growth; it was not restricted to the blood-vessels, but also involved, to some extent, the lymphatic vessels. He suggested that on careful measurement Dr. Gray would find changes in the upper limb too.

Dr. J. M. H. MACLEOD (President) said that this type of nævus, apart from the condition of the veins in the leg, was unusual; he wondered whether it was partly lymphatic, as the purple part of the lesion suggested lymphangioma circumscriptum.

He recommended repeated freezings with carbon dioxide.

Dr. A. M. H. GRAY (in reply) said he did not think this condition was hemihypertrophy, as suggested by Dr. Thomson, and he did not gather whether Dr. Thomson thought that so great a difference in the arterial circulation as there was here could come under that heading. The upper limbs of the patient would now be measured, to see whether there was a difference in the arms of the two sides.

In the section he saw nothing suggesting lymphangiomatous change; some of the patches were very flat. A section should be taken from the thicker tissue lower down and examined, before giving a definite opinion.

1 F. Parkes Weber, "Hæmangiectatic Hypertrophy of Limbs," *Brit. Journ. Child. Dis.*, London, 1918, xv, p. 13. See also C. C. Beatty, *Proc. Roy. Soc. Med.*, Section for Disease in Children, 1927, xx, p. 20; and Susman and McCredie, *Med. Journ. Australia*, Sydney, 1927, ii, p. 581.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Sir CHARLES GORDON-WATSON, K.B.E., C.M.G., F.R.C.S.

[*March* 14, 1928.]

Five Cases of Small-gut Obstruction Round Colostomies.

By W. B. GABRIEL, M.S.

IN 1919 I made a short communication to this Sub-Section regarding a case of small-gut obstruction round a colostomy [1]. I wish now to recapitulate the leading features of this case, and then to describe four others which have occurred at St. Mark's Hospital since then, together with some considerations on this variety of obstruction.

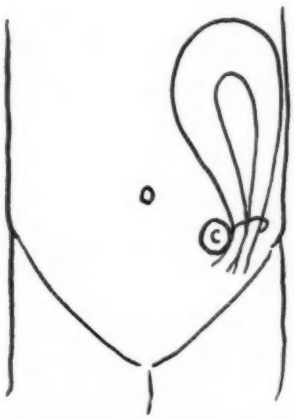

FIG. 1. Case I.—Small intestine has passed from below upwards. Operation findings three days after abdomino-perineal excision.

1.—W. D., a male aged 57 : abdomino-perineal excision of the rectum by Mr. Aslett Baldwin, May 28, 1919. A terminal left iliac colostomy was established in the usual manner. Two days later the colostomy became gangrenous, and the following day it was clear that an intestinal obstruction had developed. A further operation was performed (that is, the third day after operation) which revealed the cause of obstruction : a loop of small intestine had passed round from below upwards through the internal foramen between the colostomy and

the parietes. The obstructed loop was greatly distended and reached up to the splenic region. It could not be reduced. Mr. Baldwin, therefore, trimmed off the gangrenous part of the colon, and returned the stump into the abdomen, thus freeing the loop of small intestine. The blind end of pelvic colon was closed with purse-string sutures, and a transverse colostomy was established, with a Paul's tube tied in. The patient did not survive but succumbed the same evening.

II.—G. G., a male aged 53, was admitted into St. Mark's Hospital in July, 1920, under the care of Sir Charles Gordon-Watson. He had a small carcinoma in the lower part of the rectum, extending to the anus, with enlarged glands in both groins. Sir Charles did a gland-dissection of both groins (the left glands being subsequently found to be occupied by columnar-celled growth), and then, it being the time of the summer vacation, the case was handed over to me to complete. On August 26, 1920, I performed as a one-stage operation a left rectus colostomy, followed by a perineal excision of the rectum. I tied a catheter into the colostomy, which was opened widely next day. On August 29, a poor result was obtained from the wash-out, and the patient vomited some dark brown material. The abdomen was distended

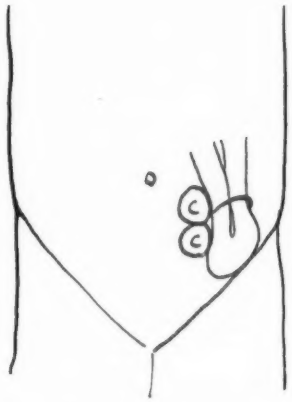

FIG. 2. Case II.—Small intestine has passed from above downwards. Operation findings three days after perineal excision and left rectus colostomy.

and tympanitic, but moved well on respiration, and was not rigid. The perineal wound was clean and dry. At 6 p.m. that day the abdomen was more distended, and at 8.30 p.m. I performed a further operation through a paramedian incision. There was an acute small-gut obstruction, which I traced to a loop of small bowel about 6 in. in length which was strangulated on the outer side of the colostomy: it had entered the foramen from above downwards. With gentle traction I reduced the loop: the bowel was viable and I closed the abdomen. At midnight the patient was cold and sweating, with irregular pulse, and he died early in the morning of August 30. A post-mortem examination was refused.

III.—F. S., a male, aged 59, was admitted into St. Mark's in November, 1924, under the care of Mr. Lockhart-Mummery. He had a growth of 15 cm., for which Mr. Lockhart-Mummery performed an abdomino-perineal excision of the rectum on December 1, 1924. The man made an excellent recovery and was discharged on January 10, 1925.

He remained quite well until February 1, 1925, when he had an attack of abdominal pain followed by vomiting, with relief. The pain recurred next day, but the colostomy acted normally on both of these days. The condition then settled down and he is recorded as having

been normal until early in the morning of February 8, when there was a sudden onset of colicky pain, which woke him up. It was griping in nature and he vomited with relief. The pain continued without cessation and on February 9 he was re-admitted to the hospital. He then looked extremely ill: tongue dry and furred; breath foul; eyes sunken; anxious appearance. The abdomen was not unduly distended; the abdominal wall was soft and no mass was palpable; the percussion note was tympanitic. When a finger was passed into the colostomy tenderness was found at this point. The perineal wound was well healed. An enema into the colostomy produced no result. I was called upon to deal with the case and operated the same evening. I explored the abdomen through a vertical incision just to the right of the mid-line, and found distended and congested small intestine. After careful search I discovered that a loop of small gut had passed down, from above downwards, on the outer side of the colostomy where it was strangulated, being acutely inflamed and very adherent. After making a transverse extension of the incision across the left rectus muscle, I succeeded in freeing the loop, but it was very friable and somewhat damaged. I resected the loop and restored the continuity by a lateral anastomosis. His condition was quite good

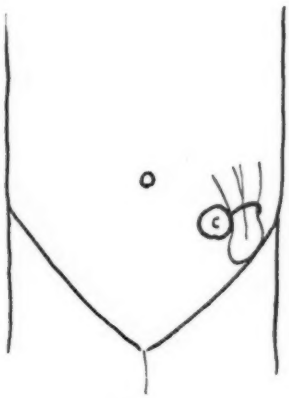

FIG. 3. Case III.—Small intestine has passed from above downwards. Operation findings two months after abdomino-perineal excision.

at the end of the operation. The following day his pulse was rapid but he was not vomiting and was sleeping well. I thought he would recover but he had a relapse on February 11 and died at 9 p.m. Post-mortem examination showed the anastomosis to be holding well: it was 3 ft. from the ileo-cæcal valve with no collapsed bowel below it. There was a large pyonephrosis of the left kidney, the pelvis being occupied by a large branched calculus with many smaller stones. The right kidney was normal. This renal trouble may have turned the scale against his recovery.

IV.—G. G., a male, aged 64, was admitted into St. Mark's, under my care, in August, 1927. He presented an ulcerating carcinoma in the lower third of the rectum which I treated by colostomy and perineal excision. The colostomy was done on August 16, under local anæsthesia, as the patient had a severe chronic bronchitis. The pelvic colon came up quite readily without any tension. Perineal excision was done on August 29: the man made an excellent recovery and returned home on October 1. He continued at home in good health with no trouble from his colostomy until November 30, when he had slight colicky pains all over the abdomen and vomited in the evening. The trouble became progressively worse,

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with pain, vomiting, and abdominal distension. No result was obtained from the colostomy wash-outs after November 30. He was not sent into the hospital until the evening of December 5. He was then obviously moribund; temperature was subnormal; he was cold and clammy and had frequent effortless vomiting of foul-smelling fæculent material. His abdomen was greatly and uniformly distended; the colostomy appeared quite normal and nothing definite could be felt either with a finger in the colostomy or elsewhere in the abdomen; the perineal wound was healed and perfectly satisfactory.

Operation at 10.30 p.m. After washing out the stomach, during which the patient nearly died on the table, I opened the abdomen under local anæsthesia: greatly distended small gut immediately presented. I rapidly performed an enterostomy and returned him to the ward. He was far too ill to attempt any sort of exploration or extensive manipulation. He died December 6 at 1.45 a.m. Post mortem, it was found that the whole of the small intestine

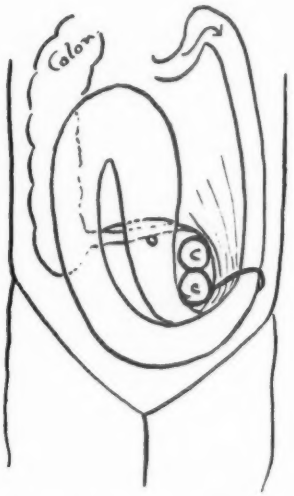

FIG. 4. Case IV.—The entire small intestine (represented as a single loop) has passed through the foramen from above downwards. The returning terminal ileum is constricted by the taut edge of the mesentery. Post-mortem findings three months after left rectus colostomy followed by perineal excision.

had passed down to the left of the colostomy. The distended loops reached round on the right of the colostomy and filled the entire abdomen. The bowel was not actually constricted in the internal foramen, but the mesentery had been dragged perfectly taut by the distension of the gut, and the efferent loop was constricted by the tight edge of the mesentery. The whole of the small intestine was greatly distended except the terminal 7 in. which was collapsed and empty. There was no thrombosis of the mesenteric vessels and no evidence of any recurrence.

V.—Mrs. E. S., aged 55, admitted under the care of Mr. L. E. C. Norbury, with an early carcinoma in the middle third of the rectum. On January 4, 1928, Mr. Norbury did a left rectus colostomy, and on January 18 he performed a perineal excision of the rectum under spinal anæsthesia, with a little gas and ether towards the end of the operation. Subsequently there was a certain amount of vomiting, but not such as to cause anxiety: the patient took fluids well by mouth and her general condition was good: her adomen was quite flat and

soft. At 7.30 p.m. January 20 the vomiting began again and the general condition began to deteriorate: tincture of iodine was given by mouth in minim doses and the vomiting ceased: the abdomen was definitely not distended. She was quiet and slept at intervals until 1 a.m. on January 21, when she became rapidly worse: her abdomen now was very slightly distended, but she quickly passed beyond the stage at which operative treatment was possible, and she died at 2 a.m.

Post-mortem examination showed that a small-gut obstruction was present 4 ft. from the duodeno-jejunal flexure. A loop 9 in. in length had passed through the foramen on the outer side of the colostomy from below upwards and was there strangulated. The bowel was not adherent and was reduced at the post-mortem by gentle manipulation from below.

In a consideration of these cases the following points may be made:—

1. *Type of Colostomy.*—A small-gut obstruction can occur round a colostomy if the foramen is not closed by suture, whether it be of the left rectus type in

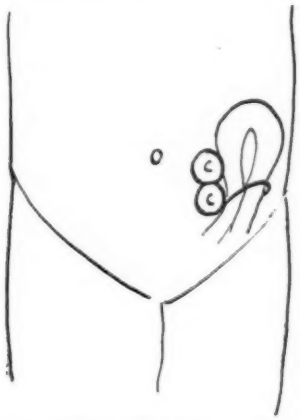

FIG. 5. Case V.—Small intestine has passed from below upwards. Post-mortem findings. Death occurred two and a half days after perineal excision with previous left rectus colostomy.

continuity, or whether it be a terminal colostomy in the left iliac region. The left iliac colostomy in continuity has not been done at my hospital as a routine in recent years, and therefore I can make no comparison between this operation and the rectus colostomy.

2. *Incidence.*—It is unfortunate that in five of our excision cases at St. Mark's Hospital since 1919 the patients should have been carried off by this complication. The two cases in which intestinal obstruction developed several months after operation are especially disquieting. The following table shows the incidence to be such that this form of obstruction certainly needs consideration.

TABLE SHOWING INCIDENCE OF SMALL-GUT OBSTRUCTION ROUND COLOSTOMIES AT ST. MARK'S HOSPITAL FROM 1919 TO JANUARY, 1928.

Operation	Total number of cases	Number of cases of obstruction and time of onset
Perineal excision with left rectus colostomy	165 ...	3 (2 immediate, 1 delayed)
Abdomino-perineal excision with terminal left iliac colostomy	25 ...	2 (1 immediate, 1 delayed)

52 Gabriel: *Five Cases of Small-gut Obstruction round Colostomies*

3. *Time of Obstruction.*—The series shows that this type of obstruction can occur as an immediate or as a delayed complication, three having occurred within three days of operation and two after intervals of two and three months respectively. An interesting fact is that both cases in which the small intestine passed from below upwards through the foramen occurred within three days of excision of the rectum. We may reasonably suspect that the actual passage of the bowel through the foramen occurred in the course of the excision operation, the patients then being in the left lateral position, head down, and with the abdominal wall relaxed by spinal anæsthesia.

4. *Mortality.*—It is quite clear that this is an extremely fatal complication, the patient not having survived in any of these five cases. The "immediate" cases, that is, those occurring a few days after operation, were particularly difficult from the point of view of diagnosis. The symptoms were very insidious and it was impossible, except at a late stage, to say that the vomiting was more than could be explained by the usual post-anæsthetic vomiting. Abdominal distension was not marked until late, when the patient's general condition deteriorated at a most alarming speed, rendering a further operation hopeless or impossible. The two "delayed" cases, occurring after discharge from hospital, had warning symptoms for days before they came in again and might have been saved by earlier operation. It is highly probable that the small gut had reached its dangerous position through the foramen for a considerable time before the final acute symptoms supervened.

Conclusions.—With the experience of these cases in mind, I should in future suspect this type of obstruction in priority in a case with obstructive symptoms after colostomy *unless* there was good reason for looking elsewhere, i.e., previous closure of the foramen, presence of secondary deposits, or definite evidence of obstruction elsewhere, such as that occurring through the pelvic floor.

In these five cases the only one with any sort of localizing sign was that with gangrene of the colostomy, and this, of course, can occur apart from this type of obstruction. One was tender on digital exploration of the colostomy, but as a method of assisting diagnosis this route of examination gave singularly negative results. In all the cases the late distension was uniform, and gave no indication as to the site of the obstruction.

In future, in dealing with such a case suspected of this type of obstruction, I should explore the abdomen *first* at the site of the foramen by an oblique incision extending downwards and forwards on the outer side of the colostomy, which would open up the anterior wall of the foramen and assist reduction of strangulated small gut. If this drew a blank, a mid-line incision could then be done.

In my previous communication in 1919, I said that so far as this artificial and potentially dangerous foramen was concerned, it might prove worth while to close it at operation by suturing the colon, as it comes forwards, to the parietes or by some other method of colopexy. I thought then that this first case might have been an isolated example of this mischance and I did not follow the matter up until the two recent cases again forcibly drew my attention to the condition; they showed clearly that this type of obstruction is a recurring danger which needs active care on our part to prevent. And it is capable of prevention. It is interesting at this point to mention the fact that as long ago as 1895 Harrison Cripps [2] described a single case of this type of obstruction following an inguinal colostomy. Case III, described in this paper, is almost exactly similar to Cripps' original case in the recurrent symptoms which occurred before the final and fatal obstruction set in.

Hitherto in this country there does not seem to have been any general recognition of this danger associated with an ordinary colostomy, and it is highly probable that in past years there may have been a steady incidence of these cases, most of them

probably having passed unrecognized. The present series of five cases does not necessarily represent the total at St. Mark's during the last nine years; there may have been others, particularly among the inoperable cases treated by palliative colostomy which may have been missed by not being explored, by lack of post-mortem examination or by not being sent in to hospital again if obstruction supervened after discharge from hospital.

The condition is mentioned by Rowlands and Turner [3], who quote Cripps' case, but no suggestion is given in regard to prevention. A recent paper by Rankin [4] is important: he reports from the Mayo Clinic their usual method of performing colostomy: the incision is a left lateral muscle-split, close to the left iliac spine; the pelvic colon is brought up—"care being taken to shut off the space between the sigmoid and the lateral parietal peritoneum by a purse-string suture to prevent the small bowel from slipping behind the sigmoid and producing obstruction." He does not mention definitely if this complication had occurred in their hands

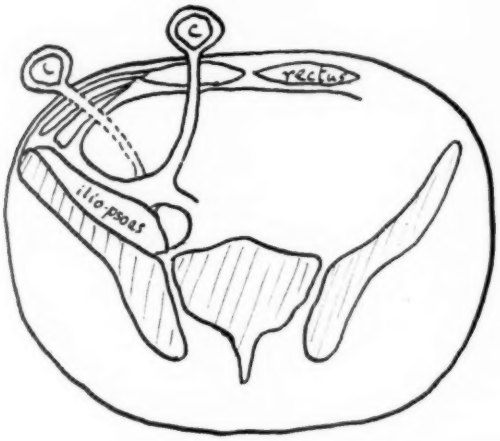

Diagram indicating the large size of the foramen produced by the rectus colostomy compared with that of the iliac colostomy.

before this manœuvre was adopted, but it is unlikely that the precaution would have been taken on theoretical grounds alone. Intestinal obstruction does not occur in the list given of the causes of their hospital mortality after excision of the rectum.

In conclusion, it should be our duty when doing a pelvic colostomy to close this internal foramen as a matter of routine; there is sufficient evidence now to prove that it constitutes a real danger when left open. For an abdomino-perineal excision I would suggest that the stab incision for the colostomy should either be enlarged or should be replaced by a formal muscle-splitting incision, with definite closure of the foramen by suture after the bowel has been brought out. For a pelvic colostomy in continuity the left iliac incision now appears to be a necessity, and I have abandoned the left rectus incision: it would be a most difficult and extensive procedure to close securely the much wider foramen left by the rectus incision (see diagram). It is only a short time since I spoke before this Sub-Section in favour of the rectus colostomy,

chiefly in regard to the lessened risk of ventral hernia formation, and it is quite certain that the rectus colostomy has many merits in the way of accessibility and control. Compared, however, with this fatal type of obstruction a ventral hernia at the site of the colostomy is a minor matter which is capable of repair by operation if it should develop to a serious degree. I have recently done this successfully in a stout subject with an enormous ventral hernia which had come through the abdominal wall just below a left iliac colostomy of six years' standing.

I am now doing all pelvic colostomies through an oblique lateral incision with closure of the foramen by a purse-string suture as described by Rankin, and I can testify to the ease and completeness with which the closure can be effected. One feels confident after doing this operation that this complication is excluded once and for all.

Finally, I wish to record my thanks to my colleagues at St. Mark's for allowing me to incorporate their cases in this paper.

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Method of Removing a Pedunculated Adenoma of the Sigmoid through the Rectum.

By Sir CHARLES GORDON-WATSON, K.B.E., C.M.G., F.R.C.S.

A PATIENT, aged 56, was under my care in July, 1923, having a pedunculated adenoma of the rectum, about the size of a walnut, situated about 4 in. from the anus. This was transfixed and removed, and was reported on by the Clinical Research Association as showing malignant changes. No recurrence has taken place.

In February, 1928, the patient came to me complaining of some looseness of the bowels with passage of slime.

On examination of the rectum there was no evidence of an adenoma, but by sigmoidoscopy a pedunculated adenoma was seen 12 in. from the anus, just beyond reach of the sigmoidoscope. In view of the past history and the microscopical report I decided to open the abdomen and investigate.

The adenoma was about one-third of the way up the sigmoid and was solitary. The patient was placed in the lithotomy position and the sigmoidoscope was passed. My assistant then grasped the adenoma through the abdominal wound, and manipulated it downwards and steadied it while I passed a snare through the sigmoidoscope over the polyp. Then by a combined pull and push the sigmoid was invaginated until the polyp could be felt with the finger in the rectum. The neck of the tumour was then clamped with long crushing forceps, the snare withdrawn and the polyp cut off. A ligature was passed and tied behind the forceps, with ends left long enough to project through the anus when the bowel was allowed to slip back. The abdomen was then closed.

On microscopical examination the polyp proved to be a simple adenoma.

This operation may seem a somewhat severe procedure for an innocent tumour, but in view of the after-history of these tumours in many instances, it is, I believe, fully justified.

The removal of such a growth after opening the pelvic colon, involves considerable risk, and has to my knowledge proved fatal. I have never attempted it; I would prefer to resect, if the method I have described failed.

The question as to what further action, if any, should be taken if a polyp so removed proved to be malignant is beyond the scope of this short communication.

Bilharzia Hæmatobia of the Rectum.

By J. P. LOCKHART-MUMMERY, F.R.C.S.

THE patient was a young officer who had been stationed in India and also on the Suez Canal. He had suffered from intermittent hæmorrhage from the rectum for some time. I made a sigmoidoscopic examination, and from the appearances—having previously seen a similar case—I came to the conclusion that the lesions were due to bilharzia parasites. There were only about six lesions altogether in the rectum and it seemed probable that the parasite was dying out and that there had never been a serious infection. The patient also had piles and it is possible the bleeding may have been mainly due to this cause. The case is shown chiefly from the point of view of diagnosis, as the condition is rare and would not be detected unless one knew what the appearance meant. As yet I have not been able to verify the presence of the parasite in the stools in this case, although I still hope to do so.

A similar case is illustrated in Dr. Bensaude's Atlas. The drawing was made from a young girl in whose stool the eggs of the bilharzia could be demonstrated. I have seen one other case.

Specimen of Villous Adenoma of Rectum.

Shown by LIONEL E. C. NORBURY, F.R.C.S.

MRS. S., aged 55. "Watery discharge" from rectum for some years: growth removed from rectum ten years and three years ago.

Examination.—Large villous growth of upper rectum (?) undergoing malignant changes.

Treatment.—Rectus colostomy followed fourteen days later by perinæal excision of rectum. Death from intestinal obstruction due to loop of small gut passing through foramen between colostomy and lateral abdominal wall.

Pathological Report.—Simple villous adenoma.

Specimen of Perforated Diverticulum of Pelvic Colon.

Shown by LIONEL E. C. NORBURY, F.R.C.S.

MALE, aged 40. Previous operation for acute intestinal obstruction by another surgeon; mid-line sub-umbilical incision—pelvic colon colostomy through this incision. Lesion present said to be inoperable carcinoma of pelvic colon, with extensive tumour formation. No spur to colostomy. After some months, great improvement in general condition. Fæces passing per rectum owing to inefficient spur.

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Examination under Anæsthetic.—No mass felt. Sigmoidoscopy showed normal rectum and lower pelvic colon. Obstruction considered to have been due to diverticulitis of pelvic colon.

Operation.—Resection of colostomy with several inches of pelvic colon: abscess cavity found in pelvis in connexion with bowel. Some 5 in. of bowel removed—end-to-end anastomosis—with temporary cæcostomy. Uninterrupted recovery.

Pathological Report.—Perforated diverticulum of pelvic colon leading into abscess cavity. No other diverticula present in the specimen.

Specimen of Carcinoma of Descending Colon Associated with Multiple Polypi of Large Bowel.

Shown by LIONEL E. C. NORBURY, F.R.C.S.

FEMALE, aged 29. No previous history of constipation—increasing difficulty with bowels for fourteen days culminating in complete intestinal obstruction with marked peristalsis.

Operation.—Mass felt in descending colon—cæcum and transverse colon much distended.

Treatment.—Temporary cæcostomy, followed at a later date by resection of the affected bowel. Lateral anastomosis between pelvic and transverse colon. Transverse colon found to be still much distended in spite of cæcostomy. Polypus found in descending colon close to growth and proved to be a simple adenoma. Several polypi removed from anastomotic stoma between pelvic and transverse colon. General polyposis of large bowel undoubtedly present.

Pathological Report.—Growth of descending colon. "Adeno-carcinoma."

Two Cases of Left Lumbar Acute Abscess caused by Carcinoma Coli.

By E. T. C. MILLIGAN, O.B.E., F.R.C.S.

I.—Male, aged 62. Pain and tenderness in left lumbar region with high fever of two weeks' duration.

Urine: Cloudy with pus. Left kidney function depressed to indigo carmine excretion.

Sigmoidoscopy: Normal. *Barium Enema and X-rays*: Normal.

Operation: Left lumbar incision; large pericolic abscess evacuated; fæcal-smelling pus.

Wound healed in four weeks.

Skiagram of chest showed secondary neoplasm of lungs.

Post-mortem examination three months later: (1) Primary carcinoma of colon just below splenic flexure; (2) Secondary carcinoma in lymph-glands, liver and lungs.

II.—Male, aged 58. Pain and tenderness in left lumbar region with high fever and wasting of three weeks' duration.

Urinary Tract: Normal. *Sigmoidoscopy*: Normal.

Operation: Left lumbar incision; fæcal-smelling pus in large quantities evacuated. Acute intestinal obstruction one week after operation. Immediate cæcostomy performed; obstruction relieved; abscess healed in three weeks.

Barium Enema and X-rays: Negative. Three weeks later, exploration of left colon by oblique abdominal incision. A constricting carcinoma of the iliac colon, firmly adherent to the iliacus muscle, was excised and end-to-end union of bowel performed [specimen shown].

Comments.

(1) Left lumbar abscess was the first clinical condition appearing in these two patients suffering from carcinoma of the left side of the colon.

(2) The abscess in both cases healed completely after evacuation.

(3) The fæcal odour of the pus suggested its origin from the bowel rather than from the urinary tract.

(4) In both cases clinical means failed to diagnose the nature and site of the carcinoma in the bowel. The barium enema gave no help.

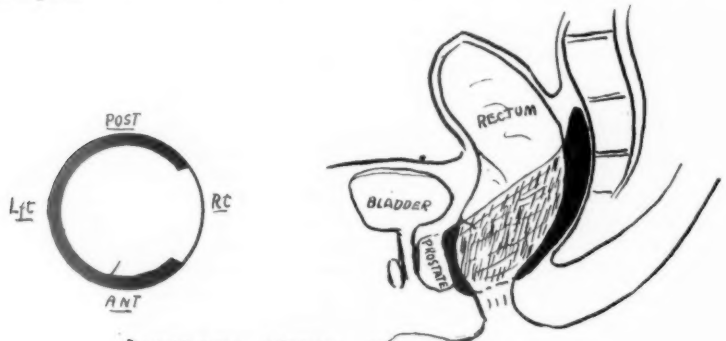
(5) It is suggested that abdominal exploration should follow the healing of such fæcal-smelling left lumbar abscesses, even if clinical methods fail to diagnose a lesion in the bowel.

(6) In one case reported resection of the carcinoma was possible after the infection had subsided.

Case of Gangrene of the Rectum following Long-standing Prolapsing Hæmorrhoids.

By E. T. C. MILLIGAN, O.B.E., F.R.C.S.

MALE, aged 53. Infective gangrene caused sloughing of the whole thickness of the rectal wall, exposing the sacrum and coccyx behind as high as the examining finger could reach, and the posterior surface of the prostate in front. The circumference of the rectal wall was destroyed, except a strip about 1½ in. wide on the right lateral aspect.



DIAGRAMS SHOWING EXTENT OF GANGRENE (BLACK).

Colostomy was performed and, after a long illness, the patient recovered.

Owing to the strip of undamaged rectal wall no stricture has developed and the colostomy can now be closed.

There still remains a small granulating area in the rectum.

The case is of interest because infective gangrene of the rectum is uncommon. Gangrene is rarely so extensive, and such a good recovery is unusual.

58 Maynard Heath : *Growth in Anus : Case for Diagnosis*

Growth in Anus: Case for Diagnosis.

By P. MAYNARD HEATH, M.S.

L. P., MALE, aged 34, was first seen in January, 1927. At that time he was said to have had piles and itching for twelve months. Examination revealed a warty growth at the anterior margin of the anus. This was removed by operation, and in the middle of it a small fistula was discovered; this was laid open and the raw surface was lightly cauterized. The pathological report on the growth removed stated that it was hypertrophied stratified epithelium.

The wound healed and the patient remained well for six months; there was then, he states, a recurrence of growth and moisture. He did not come for examination until February, 1928.

Opinions are asked as to the nature of the condition and treatment suggested.

The blood Wassermann reaction is negative.

Section of Tropical Diseases and Parasitology.

President—Dr. R. T. Leiper, F.R.S.

[*March* 1, 1928.]

Health Lessons from Bermuda.

By Andrew Balfour, C.B., C.M.G., M.D.

So far as I am aware, no scientific observations have been made regarding physiological changes induced by the Bermudian climate. Such a study would be of considerable interest, for, in Bermuda, disturbing factors encountered in the tropics can be eliminated. One of these, at least in the case of coloured populations, is ankylostomiasis. In Bermuda the hookworm is not in evidence and the view commonly held is that the porous soil, which dries rapidly after rain, is not favourable for the development of the larvæ, for which a certain degree of moisture is required. Yet in scientific work nothing must be taken for granted. Possibly the fact that few, if any, of the inhabitants go unshod may explain in large measure the immunity from one of the most widespread and debilitating of parasitic diseases. A lesson, apparently paradoxical, is here inculcated: the value of carrying out research upon diseases in countries where such diseases do not exist. Negative evidence has a value of its own. Another point of interest is the apparent disappearance of the chigger flea from Bermuda. Fifty years ago it was well known there and references to it and to the trouble the female flea causes by making her accouchement chamber in the human skin, are not infrequent in descriptions of the Colony. Here again it is possible that the more general use of shoes has ousted the intruder, but if so, one would expect that it might have found another host, namely the pig, which is common in the island and which, in other countries where the chigger flourishes, is frequently victimized. Is it then conceivable that this tiresome flea has encountered some adversary whose onslaughts have caused its disappearance? We cannot say, for research is lacking and we draw the lesson of the great need of a laboratory and of skilled inquirers even in a tiny territory where the death-rate is comparatively low and disease not greatly in evidence.

A similar conclusion is reached when we find that malaria and anophelines are absent. In this respect Bermuda comes into line with the Seychelles, Fiji, Samoa and other islands of the sea. It is certainly remarkable that anophelines have never gained a footing in the Colony, for it possesses numerous marshy tracts which appear in every way to be ideal breeding grounds for those species of mosquito which serve as vectors of malaria. Furthermore Bermuda has for several hundred years been in close communication with places where anophelines abound, as, for example, the eastern seaboard of the United States and the West Indies. The problem certainly demands investigation, and useful lessons might be learned by a properly conducted survey. Can the reason for the immunity as regards anophelines be explained by the hydrogen-ion content of the waters of the Bermudian swamps? MacGregor, I think, first directed attention to the important rôle of the reaction of waters in determining whether or not they are suitable nurseries for mosquitoes. He commenced his observations in Surrey and continued them in Mauritius, where he found that the local anophelines were never present in water with a pH below 7·4 and only rarely when the pH was as low as 7·4. pH 7·4 represents a point just below neutrality on the acid side. Waters with a pH of from 8·4 to 9·5, in other words alkaline waters, usually harboured considerable numbers of

vigorously developing larvæ. Hence it would seem (and Miss Russell's work in Macedonia is confirmatory) that some anophelines will not breed in distinctly acid waters. On the other hand, observations by Senior White, Barber and Komp, and Walton and Rees Wright are at variance with MacGregor's finding. The reaction of the marsh waters of Bermuda has never been fully tested, though Major Langrishe, R.A.M.C., has conducted inquiries into the salubrity of the marine swamps with reference to the development of salt marsh mosquitoes and has found pupation occurring in water with a chlorine content as high as 1,500 parts per 100,000, i.e., about 75 per cent. sea water. This, however, by the way.

Another factor requiring consideration in connexion with the absence of anophelines in Bermuda is the part which larvivorous fish may have played. There exists in all the marine swamps and probably in most of the inland waters, a species of fundulus identified in the United States and named there as *Fundulus bermudæ*. This little fish occurs in great numbers and feeds voraciously on such mosquito larvæ as are present, namely those of *Culex fatigans*, the common brown gnat of the tropics and sub-tropics, and those of the *Aëdes sollicitans* and *A. tæniorhynchus*, the two species of salt-marsh mosquito present in the Colony. These latter were in all probability introduced from the eastern American seaboard, where they are common in many localities, notably in New Jersey. Now, as so frequently happens, a balance has been struck between the local fundulus and the mosquito larvæ on which it preys. The latter find protection from aquatic vegetation which prevents the fish getting at them. Indeed, in one swamp pool I took with three successive dips, mosquito larvæ, a small "mullet," (as the fundulus is called), a dragonfly larva and several notonectidæ. The fish, the dragon-fly larvæ and the water-boatmen are all inimical to mosquito larvæ, but could not get access to them owing to the presence of weed. It is, however, a reasonable hypothesis that anophelines have been introduced, that the females have laid their eggs, and that these have turned into larvæ, but that the broods were wholly exterminated by the local fish before any imagines emerged from pupal cases.

Another, less credible, is concerned with the presence or absence of aquatic plants of the natural order Characeæ in Bermudian waters. Caballero, in Spain, declared that certain species of the genus *Chara* are highly inimical to anopheline larvæ, apparently because the plants yield to the water in which they grow some toxic substance which kills the larvæ. In certain countries Caballero's findings have been confirmed. Accordingly I searched for chara in the large Pembroke swamp near Hamilton, the capital. I could find no specimens, perhaps because it was the winter season, but there is a botanical record to the effect that a species known as *C. foliolosa* has been taken in that very marsh. This species, however, is said not to be detrimental to anopheline larvæ in other parts of the world, but actually to afford them shelter from their enemies.

Hence we need not consider a botanical cause as explaining the colony's freedom from spotted-winged mosquitoes. But we may learn a lesson, not from Bermuda, but from what has recently happened in Barbadoes, that to view the immunity of Bermuda with complacence may precipitate a calamity like that which has befallen Barbadoes, though we must admit that the danger is not so obvious.

Amongst the mosquitoes of Bermuda is *A. ægypti*, better known under the pseudonym of *Stegomyia fasciata*, the vector of yellow fever.

The history of yellow fever in Bermuda teaches many lessons, both medical and sanitary. There seems to have been a bad outbreak in 1699, there was certainly a severe epidemic in 1796, the year in which New York and Philadelphia were scourged by the malady, but there was no further prevalence until 1812 when the garrison and dockyard were decimated. In 1818 and 1819 the fever was again in evidence a nd there appear to have been "dropping" cases for several years afterwards. In 1837,

H.M.S. *Seringapatam* introduced the disease from Havana, and in 1843, when South Carolina and other parts of the United States were scourged, the Colony was not exempt. The epidemic was severe and occasioned many deaths, especially amongst the soldiers and the convicts on the hulks. The succeeding epidemics of 1853, 1856, and 1864 are better known, and on several occasions when in Bermuda in 1923 I met persons who, as children, had been ill with yellow fever in 1864. It would seem that, subsequent to that year and possibly even down to the earlier "seventies," occasional cases of yellow fever were recorded, but there was nothing in the nature of an outbreak. Since those days the islands have been free and we are led to ask why such an immunity has been vouchsafed them, considering that the mosquito vector is still present in abundance, and indeed that, until comparatively recently, no serious measures were taken to combat it. I am inclined to think that the substitution of steamers for sailing ships has had something to do with the matter, for, as is well known, *A. ægypti* found the old-fashioned wooden vessel a much more suitable habitat than the iron tank of the present day. The ousting of the sailing ship is, however, insufficient to account wholly for the immunity of Bermuda from yellow fever. Probably the fact that the disease never gained a *permanent* footing in the colony is one reason and the failure of the parasite to become established there is itself to be explained by the winter season being unfavourable to it.

Indeed, it would seem possible to say which of the numerous fever outbreaks in Bermuda in olden days were, and which were not, examples of "yellow jack" by considering whether they were limited to the summer or continued throughout the colder months. Thus the epidemic of 1779-80, which persisted throughout the so-called winter, was almost certainly typhus, as indeed the name of "gaol fever," which was applied to it, seems to indicate. It should be noted that this occurred during a war period as did the undoubted typhus fever of 1867.

Another factor which, partly owing to the successful crusade of the Americans, has doubtless been operative of late years, is the stamping out of yellow fever from many of its old-time endemic foci in the new world. There was, however, in 1921, a small epidemic in British Honduras, a country with which Bermuda is in direct steamship communication. It is therefore unlikely that the long immunity enjoyed by the Colony depends chiefly on what is called "chance," a concatenation of circumstances on which, in matters sanitary, man is apt to rely to his ultimate undoing. Barbadoes did so and escaped yellow fever from 1881 to 1907, but retribution fell upon it in 1907, and from 1907 to 1909, at least 100 persons went down with the disease, of whom thirty-six died.

In connexion with the history of yellow fever in Bermuda a curious lesson may be learned, happily one which very rarely has to be impressed on the medical man. It is that, if a physician desires effectively to put his medical knowledge to base uses, he must first be certain that such knowledge is accurate. In 1864, during the American Civil War, a diabolical plot was hatched in the Islands by one Blackburn, a physician, hailing, it was said, from New Orleans. Its object was the collection of blankets, sheets, underwear and handkerchiefs stained with black vomit, together with poultices from fever cases, for the purpose of infecting New York and other cities of the Northern States.

In 1900, Reed and his colleagues incriminated the mosquito. Yet long before this date the mosquito had been suspected and if previous to the ast epidemics of yellow fever in Bermuda, the hypotheses advanced by Blair of British Guiana, and still more by the famous Beauperthuy, the French Creole, had been broadcasted, it is conceivable that they might have been adopted and measures might have been taken to protect against mosquitoes and so to stay the plague. In those days, however, the literature on tropical medicine, though plentiful, was not easily

obtainable. Something is to be gathered from the methods now in force for dealing with *A. ægypti*. In Bermuda, water for domestic uses is stored in large stone tanks. Every house is supposed to have a tank and these form ideal nurseries for the stegomyia broods. The most effectual way of dealing with the larvæ is by placing goldfish or the local fundulus in the water. The fish thrive and keep the water from mosquito larvæ unless, and here a lesson obtrudes itself, the tanks are not periodically cleaned and a growth of aquatic vegetation is permitted to accumulate on their sides. If this occurs the fish may find a diet more to their liking than the "wig-wags"—the vulgar name applied to all mosquito larvæ in Bermuda.

Here, as has been demonstrated elsewhere, the presence of fish in water stored for drinking purposes is in no way deleterious. Houston (London) showed that roach, dace, perch and other species of fish yield to water, *B. coli* both in its typical form, that of Escherich, and in allied forms. He regards the organisms as being probably quite harmless. On the other hand, Havens and Dehler in the United States found that the top minnow, *Gambusia affinis*, yields *B. pyocyaneus* to water but not *B. coli*. The former organism was observed indeed to have an inhibitory effect upon the latter, so much so that considerable numbers of top minnows may render the *B. coli* index an unreliable guide to pollution. Here surely further research is required, and nowhere could the investigation have been better conducted than in Bermuda if only laboratory facilities had existed there. I am glad to say that in 1925 a bacteriological laboratory was established at the King Edward Memorial Hospital.

Yellow fever, clinically and pathologically, closely resembles spirochætosis icterohæmorrhagica. In the past, and indeed until quite recently, the two maladies have probably not been properly differentiated in America, and from the work of Schüffner and Mochtar, Theiler and Sellards, of Gay and Sellards, Mosoya and Stefanopoulo, and Puntoni there seems good reason to believe that *Leptospira icteroides*, the reputed parasite of yellow fever on the American Continent, is identical with *L. icterohæmorrhagiæ*, the organism of Weil's disease. Indeed, the recent researches of Schüffner working in conjunction with Mochtar, Proehoeman and Honig, appear to have definitely settled this question in the affirmative. In any case, it is of interest to determine, in countries where yellow fever occurs, or has occurred, whether the rat populations harbour the second of these spirochætes. This has not been done in Bermuda, but the research, a simple one, might with advantage be undertaken. I made the suggestion at the time of my visit but am not aware that any steps have been taken to give effect to the recommendation.

In olden days the graves of those who had died of yellow fever were regarded with fear and suspicion. It was believed that they gave forth dangerous emanations, and cemeteries were frequently cited as places where infection might be contracted. We smile at these beliefs to-day, and yet graveyards did play an important part in the ætiology of yellow fever and might do so again in Bermuda. Nearly every cemetery is a prolific breeding place of *A. ægypti*, the water stages being found in the water contained in the different kinds of flower receptacles on the graves and monuments. Surely a lesson may be derived from a slab bearing the names of several members of a family wiped out by yellow fever and with the pious words graven upon it: "God's will be done," yet also bearing a jam jar full of flowers and full also of stegomyia eggs, larvæ and pupæ!

It is of interest to note that the Bermudian is more or less immune to the bites even of such vicious and irritating mosquitoes as the salt-marsh breeders, This fact was observed long ago and is recorded by a certain Susette Lloyd who produced a book about Bermuda in 1835. No one, so far as I know, has ever worked out the mechanism of this type of immunity. Schaudinn asserted that the pain and discom-

fort of the mosquito bite were due to the discharge into the wound of yeasts derived from the œsophageal diverticula, and Bruck, in an investigation, far from precise, obtained from the organs of culex an urticaria-producing substance which he termed "culicin," but the nature of the irritating substance—and indeed the whole question—require further exploration even though the work of Roy in India and that of Pulido in Spain have added somewhat to our knowledge. The interesting research of Yorke and Macfie on the action of the salivary secretion of mosquitoes does not deal with this aspect of the question.

A lesson in human credulity and ignorance, those factors which more than any others hinder sanitary progress, can readily be drawn from Bermuda. When collecting the larvæ of *C. fatigans* in one of the island marshes I was accosted by a comely black wench as follows: "What you doing? You trying to get rid of mosquitoes Sah? You never do that. Mosquitoes are the gift of the good God to Bermuda." If this cheerful and resigned person's view was correct, she taught me another lesson, that of simplification of the mosquito nomenclature in the Colony, for why not class the four species of Bermudian culicines under the single generic name "dorothea" and call them all *Dorothea bermudiensis*? Yet I venture to doubt her assertion. It is, at least, hard to reconcile it with certain happenings at St. John's Church in Pembroke Parish, recorded by its vicar in a pastoral letter dated October, 1923. He wrote as follows: "For the past few Sundays an experiment has been tried and has proved successful in driving out the mosquitoes from Pembroke Church. The evening congregation can now worship in some degree of comfort. If there are any persons who have absented themselves because of the attack of the mosquitoes, I would ask them to make another trial. We want your help to build up this service." If mosquitoes are God's gift to Bermuda, how explain these onslaughts upon the faithful in the sanctuary? As a matter of fact the worthy vicar unwittingly provided a lesson as to the danger of lack of thoroughness in anti-mosquito operations, for a large number of his winged foes were due to flower receptacles on the graves surrounding the place of worship.

Mosquitoes suggest water, and in the tropics and sub-tropics water, unfortunately, often suggests dysentery. Dysentery is rare in Bermuda, probably because there is no surface running water liable to gross pollution. Bermuda, however, abounds in flies. Fly-breeding indeed might, at the time of my visit, have been regarded as one of its principal industries, largely owing to the facts: (1) that the horse-drawn vehicle still persists in Bermuda as, apart from the push bicycle, the sole means of wheeled transport; (2) owing to the necessity of catering for the needs of tourists, the number of horses is out of all proportion to the size of the island; and (3) the large livery stables are almost invariably most insanitary. Hence *Musca domestica* and other allied diptera were greatly in evidence. I am glad to say that, thanks to good work carried out by Dr. G. B. Mason and others, matters have greatly improved in this respect. There are those who regard the fly as an important agent of infection both in amœbic and bacillary dysentery, while others consider its rôle in these conditions has been much overrated. Experiences in Bermuda support the latter opinion. The island teaches the old lesson, "pure water, little flux."

On the other hand, the fly is believed to have a good deal to do with the prevalence of enteric fever which, next to tuberculosis, is the most important communicable disease in the Colony. This is no wonder, for the conditions governing the collection, removal and disposal of excreta and refuse were far from satisfactory and still, I understand, require close attention. A lesson in the value of sanitary discipline and efficiency is to be learned from the fact that, in 1922, fifty-two cases of enteric fever were reported amongst the civilian population, thirty-eight of which were confirmed either bacteriologically or clinically, while not a single case occurred

amongst British troops. The numbers concerned, of course, were very different, but on the other hand, amongst the soldiers there was proportionately a greater number of persons at susceptible ages.

It must not be forgotten that the soldiers were protected by the triple vaccine, but Colonel Houghton, the Senior Medical Officer in 1923, informed me that, in his opinion, the immunity was in part, at least, due to the presence of a piped water supply, of a water-carriage system of sewage disposal, and to the stringent measures taken to guard against fly infection.

Antityphoid vaccination is employed amongst the civilian population at present, for the protection of contacts, but there is a growing tendency in Bermuda, as elsewhere, to rely upon it to the exclusion of other means of defence. This, I believe, is a lesson in how *not* to proceed. I am not decrying the value of anti-typhoid inoculation, which has been proved time and again, especially, as far as civil communities are concerned, in Fiji, the Virgin Islands, and amongst the Maoris of New Zealand. In times of emergency, and under certain other conditions, it has its place in civil life just as it has in time of war, but it would be a pity if it were to usurp the first place in the line of defence. There is a distinct danger that this may occur and that thereby communities will be led to neglect their obvious duties in the way of providing cleanly surroundings and carrying out essential sanitary works. It is no doubt cheaper to pay bi-annually for two preventive doses of vaccine than to be mulcted heavily in the rates for a new and satisfactory water supply, but it is not wiser in the long run. Enteric fever is only one of several filth diseases, and if a community is to live healthily, it must pay for those services which abolish filth and its results. It is not a sound sanitary policy to rely on a large scale upon a measure like preventive inoculation if only because the enteric fevers cannot, so far as ætiology is concerned, be divorced from other dangerous maladies against which an antityphoid vaccine conveys no protection. I have mentioned tuberculosis as the chief communicable disease in the Colony. Bermuda teaches the old lesson that climate has little, if anything, to do with the incidence of the "white plague." There, it is a question, in part, of housing conditions, in greater part of faulty habits and ignorance. Bermuda is one of the most thickly populated islands in the world, yet there is not much overcrowding, even though considerable stretches still remain untenanted. This is due to the fact that outside the two towns, Hamilton and St. George, almost every household occupies a detached stone-built cottage, generally standing in its own garden. Even in the towns there are few slums and practically no rookeries; indeed the middle and lower classes are probably better housed than in any other part of the world. Yet tuberculosis is rife amongst the coloured community because the value of the open window is not appreciated, and because expectoration is freely practised. The milk supply is also in some measure to blame.

Previous to my visit there had been no medical inspection of school children. This should be introduced, as thereby cases of incipient tuberculosis in the young would be detected and remedial and preventive measures could be put in force. I may say in passing that while the climate of Bermuda is admirably suited for most asthmatic and bronchitic cases, it is not altogether favourable for patients suffering from pulmonary tuberculosis, on account of the number of windy days and certain other factors. The Colony has an infantile mortality-rate of about 160 per 1,000, a sad tribute to the lack of mothercraft and the prevalence of insanitary conditions amongst the coloured population. Bermuda is systematically putting its future labour underground. Seemingly the view taken by the coloured section of the community may be expressed by the phrase "easily come, lightly go, quickly replaced." Such an attitude is alike callous and short-sighted. It is wasteful and

contrary to the teachings of hygiene and the natural instincts of humanity. If the population, or any section of it, is increasing unduly, a remedy may perhaps be found in birth-control, which is now, if properly safeguarded, regarded by the majority of hygienists as a legitimate and harmless measure. To bring children into the world merely to see them perish and to be at the expense of burying them, is surely a fatuous procedure when the means exist for safeguarding young lives and when we possess all the knowledge requisite for combating ignorance and carelessness and their dire effects.

One of the last lessons I am seeking to draw from one of the oldest and smallest of British possessions is concerned with the effect of economic developments upon the medical and sanitary life of its inhabitants. Nowadays we are a little apt to forget how profoundly industrialism altered the hygienic situation in this country, the fine yeoman population being herded into towns and, as the towns became foul, losing much of their robustness and physique. In Bermuda, especially in recent years, a change has occurred similar to this in some respects but in others wholly dissimilar.

The motive power has been provided by the dollars of the American tourists who flock to Bermuda in thousands, both in winter and summer. The towns have not greatly altered; Hamilton and St. George remain much as they have been, but insanitary conditions have sprung up in the immediate vicinity of both, and there has been a concentration of population outside Hamilton. I cannot deal with all the influences at work, or with all the changes that have occurred, but I may briefly comment on the fact that a large proportion of the population has given up tillage of the land, that it no longer produces vegetables for its own use, and has come to rely on "canned food." From a sanitary standpoint the result has been deplorable, for in almost every part of the island there is a litter of empty—or partly empty—tins providing nurseries for mosquitoes and refectories for flies, while in many places there are dumps where, mingled with refuse of various kinds, tins of all shapes and sizes disfigure the landscape. I could not, however, discover that ill results had followed a dietary largely restricted to preserved foods. There was no evidence of the prescorbutic or the scorbutic state, rickets is rare, pellagra unknown. There are undoubtedly dietetic lessons to be learned in Bermuda. But, as I trust has been apparent, the tiny Colony is full of lessons. The pity is that so few of them have been mastered, and that most of them have not been laid to heart, although in certain directions, notably as regards quarantine, Bermuda has done well. Given an enlightened sanitary policy on sound and energetic lines, the "still vexed Bermoothes" as Shakespeare called these islands, might soon boast the lowest death-rate in the world, and, what is better than a surprisingly low death-rate, a population free from the damage wrought by disease. The place has been greatly favoured by nature. It has in certain respects been spoiled and injured by man's apathy, folly, ignorance and selfishness. Yet there is hope for the future because Bermuda cannot afford nowadays to run risks and has in some measure become alive to this fact. The Colony is almost wholly dependent on the tourist traffic, and tourists and epidemics are not compatible. Hence there, as elsewhere, the old sanitary lesson is forthcoming, namely, that the chief factors concerned with hygienic revolutions are fear, discomfort, and the pinch of the pocket.

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Discussion.—Dr. G. CARMICHAEL LOW said that, judging from Dr. Balfour's pictures of Bermuda, little seemed to have been learned from the pioneer work of many years ago. Flies and mosquitoes abounded and sanitation was in a very backward state. Anopheles might be introduced there as they have been in Barbados and malaria be added to the diseases already existing.

It was of little use to send out experts to report on those islands unless their recommendations were to be carried out. With the advent of the new School of Hygiene, it was to be hoped that a better type of sanitary inspector would be sent out, but here again, unless he was backed up by the local governments, his efforts would be in vain. It is sad to think that we had British colonies at the present day as backward in their mosquito sanitation as thirty years ago.

With regard to Dr. Balfour's suggestion of examining the rats in the different islands for the *Leptospira icterohæmorrhagiæ*, this certainly should be done, as it was an important point to settle whether the epidemics which used to sweep the West Indies were all due to yellow fever. The history of most of these islands was a long immunity from the latter disease, even up to thirty and forty years, and this tended to show that it was real yellow fever and not infectious jaundice. If the latter disease had been responsible, cases should have kept cropping up during these periods. This was a piece of research work urgently requiring solution.

Dr. H. H. SCOTT: The paper is full of facts, but, as established facts, they do not lend themselves to much in the way of debate and the application of them to colonies other than Bermuda does not come within the scope of this discussion.

When I heard what was to be the subject of this evening's debate I tried to find out something of the medical history of Bermuda. All the authorities seem to make special note of the rarity of malaria, and that this must be ascribed to the absence of anopheles is supported by the fact that cases have been imported again and again, especially from the West Indies,

and had the vector been present and thriving, these cases would certainly have constituted foci for spreading the disease. Seeing that mosquitoes can be transported (aëdes, for example, at least) from one place to another and remain virulent, it is a matter for wonder that the anopheles has not propagated in a country rich in marshy sites; it seems impossible that they have never been introduced and we are driven to the conclusion that conditions inimical to them exist in Bermuda.

To take another disease mentioned: I found it stated that a typhoid epidemic occurred in 1866, mentioned in the British Army Reports. Both the civilian population and the troops suffered. Three years later there was another outbreak of forty-three cases confined to a single regiment, 710 strong, and nine of them died. The civilians appear to have escaped this time—the reverse of what Dr. Balfour mentions as of more recent occurrence.

Again, the *Lancet* of 1869 records an interesting small outbreak of yellow fever, interesting as showing that the mosquito retains infection for a considerable time, and also as an example of the belief in a yellow fever poison clinging to rooms in which cases had been nursed. "The naval authorities," runs the report, "had told off a number of sailors to clean out the quarantine hospital and the work had just been begun when a ship arrived from the West Indies with yellow fever cases on board." The sick were taken to hospital and the cleaning temporarily stopped. When the patients had sufficiently recovered (it is not stated how many recovered) the vessel left for Halifax, and twenty-seven days after the last convalescent had left the hospital, "the sailors, eight in number, resumed the work of cleaning and within the next eight days two of them sickened and died; a third fell ill six days later; the remaining five were therefore sent to Halifax under the charge of a surgeon who himself took the fever and died."

The Bermudas certainly seem to be unafflicted by some of the scourges of other tropical countries and, apart from the fact—an important one, I grant—that the sanitation is not up to Dr. Balfour's standard, would serve as a health resort; indeed, Bermuda was so regarded by ague patients nearly a century ago, and is apparently similarly regarded at the present time in spite of these drawbacks.

Dr. MANSON-BAHR said that negative evidence was of great value and much information could be obtained from the absence of diseases from isolated island groups. It did not always pay to "rub in" the sanitary errors of countries possessing manners and habits other than our own. He was much impressed at the astonishment with which Egyptian and Indian visitors regarded the lack of sanitation in some of the back streets of our great metropolis. Sir Patrick Manson had often said that to overcome the technical difficulties in sanitary administration in tropical countries the appointment of outstanding medical men as governors was most desirable. In the past some doctors had excelled as administrators, for example, the late Sir William Macgregor.

JOINT DISCUSSION No. 7.

War Section and Section of Otology.

[*April* 13, 1928.]

DISCUSSION ON THE EFFECTS OF MIDDLE-EAR DISEASE ON EFFICIENCY IN CIVIL AND MILITARY LIFE.

Chairman—Group-Captain HARDY V. WELLS, C.B.E., R.A.F.

Wing-Commander DAVID RANKEN.

ABSTRACT.—(1) Introductory.—(2) Symptoms and complications of middle-ear disease and their effects on efficiency.—(3) Service views on the disposal of men suffering from middle-ear disease.—(4) Comparison of the disability caused by middle-ear disease in civil and military life.—(5) Are the aural recruiting and invaliding standards of to-day too exacting?—(6) Aural disease in recruiting and invaliding—tables.—(7) Advantages of rigorous standards of recruiting.—(8) Attainment of aural efficiency in the Services.—(9) Is any relaxation of standards ever justifiable?—(10) Importance of civil and military co-operation.

THE subject of our joint discussion is one of considerable importance to Members both of the War Section and the Section of Otology; all Medical Officers have from time to time to consider the fitness for service of soldiers or sailors suffering from middle-ear disease; all otologists, on the other hand, are asked some time or other to state the degree of disablement from this disease in order that compensation or a pension may be rightly awarded; those who, like myself, are both Service medical officers and otologists have a dual responsibility embracing both the aforesaid decisions.

At the outset, we must recognize that the effects of any disease on efficiency will vary according to the nature and extent of the disease as also on the rapidity of cure; "middle-ear disease" is rather a loose term based entirely on anatomical considerations and including various pathological entities; these, however, fortunately for our purpose, cause much the same kind of symptoms, and diminished efficiency will be in direct proportion to the severity of these symptoms.

We are all familiar with the possible and frequent complications of middle-ear disease; recurrent earache, hardness of hearing, otorrhœa, tinnitus and vertigo form an unpleasant list; we must also take into consideration the effects of such symptoms on the general health of the individual, and in a small number of cases the danger of intracranial complications.

In my experience, hardness of hearing is by far the most frequent symptom of which complaint is made; recruits with poor acuity of hearing are unsatisfactory; their hearing varies from time to time, especially when they have a cold; they are frequently accused of inattention and complain that owing to this they are passed over for promotion; failure to hear or rightly interpret orders leads to accusations of stupidity; hence, despondency and a desire to get out of a Service in which they feel they are not likely to succeed.

The amount of disablement due to earache, tinnitus and vertigo will vary according to their severity; it is clear that a man liable to vertiginous attacks cannot be employed on duties where its occurrence might entail bodily danger; should mastoiditis or an intracranial complication arise, a soldier may be weeks or months off-duty occupying a bed in hospital at considerable expense to the State; finally, such symptoms or complications are sure to have a bad effect on the general health and nervous system of the soldier; some of them are difficult to prove or disprove and opportunities for malingering are great.

There are of course certain aspects of military life where middle-ear disease may mean not merely a disability, but a catastrophe; a pilot who cannot hear telephones

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or wireless with certainty is severely handicapped in the air; flying officers liable to unilateral or bilateral Eustachian obstruction may develop, especially when descending from a height, such severe middle-ear and labyrinthine symptoms that they lose control of their machines.

In the Services there are, broadly speaking, two distinct views as to the disposal of men suffering from intractable middle-ear disease; some consider that such people should be invalided out; others with less drastic ideas hold that this is wrong in view of the wastage of money and man-power entailed by invaliding trained or partially trained soldiers; they contend that it is better to continue to try and cure such men, or, at all events, to patch them up by treatment, when the severity of the symptoms becomes a disability; they also argue with considerable reason that many individuals carry on fairly well in civil life although suffering from chronic middle-ear disease and that the analogy should hold good for the Services.

There are however complicating factors that distinguish civil from military life; for some of the general duties of a soldier, such as sentry-go, good hearing is essential; in addition he requires a great deal of verbal instruction. Civilians however are not required to act as sentries and those who are deaf drift into occupations where acuity of hearing is non-essential; a second distinction lies in the fact that all soldiers must be able to perform their duties efficiently both at home and abroad; they must always be fit for field service and are liable to exposure to cold and wet and to epidemics of influenza or measles, all of which may cause or restart aural inflammation; finally they may be in a place where a competent aurist is not available; a civilian, on the other hand, is perfectly free to choose his own environment.

I will now pass to what I feel is the most important item of our discussion from a military point of view: Is the aural standard of recruits to-day too exacting and if so what more rational formula can be suggested? The following tables show the loss of man power entailed in the Royal Air Force during the last five years by the rejection of recruits and the invaliding out of men suffering from aural disease.

TABLE I.—RECRUITS. REJECTIONS, 1921-26.

Cause of rejection	1921	1922	1923	1924	1925	1926	Total 1922-26
Aural disease	not available	208	453	218	195	14	1,088
All others	not available	4,308	4,482	2,629	1,899	244	13,562
Total	not available	4,516	4,935	2,847	2,094	258	14,650
Aural disease percentage of total rejections	—	*4·6*	*9·2*	*7·7*	*9·3*	*5·4*	*7·4*

TABLE II.—FINAL INVALIDING OF AURAL CASES BY "LENGTH OF SERVICE GROUPS," COMPARED WITH TOTAL NUMBER OF INVALIDS OF "ALL OTHER DISABILITIES."

Service	1921	1922	1923	1924	1925	1926
(1) Less than 6 months ...	1	2	1	2	—	—
(2) 6 months to 1 year ..	6	11	2	6	2	3
(3) Less than 2 years (includes 1 and 2) ...	8	20	8	10	5	5
(4) 2 years to 4 years ...	9	19	17	15	7	10
(5) 4 ,, 6 ,, ...	2	5	11	9	14	9
(6) 6 ,, 8 ,, ...	—	3	—	—	3	10
(7) 8 ,, 10 ,, ...	—	—	1	2	1	1
(8) 10 ,, 12 ,, ...	—	—	—	—	—	—
Total (3 to 8) ...	19	47	37	36	30	35
All other disabilities ...	239	390	342	264	277	205
Aural disease percentage of total disabilities ...	*7·4*	*10·8*	*9·8*	*12·0*	*9·8*	*14·6*

TABLE III.—INVALIDING FROM ABROAD.

	1921			1922			1923			1924			1925			1926		
	Total invalided	Returned to duty in U.K.	Finally invalided	Total invalided	Returned to duty in U.K.	Finally invalided	Total invalided	Returned to duty in U.K.	Finally invalided	Total invalided	Returned to duty in U.K.	Finally invalided	Total invalided	Returned to duty in U.K.	Finally invalided	Total invalided	Returned to duty in U.K.	Finally invalided
Aural disease	2	—	2	5	3	2	8	5	2	11	6	4	17	6	6	7	4	9
All other disabilities	165	128	37	155	96	59	227	186	42	157	102	56	238	181	62	140	116	18
Total ...	167	128	39	160	99	61	235	191	44	168	108	60	255	187	68	147	120	27

NOTE.—It will be observed the disposal, i.e., "to duty" or "finally invalided" in the case of aural disease does not agree with the total invalided to U.K. for each of the years 1923-1926. This is due to the case being disposed of in a subsequent year.

It is my personal opinion, founded on my experience as an otologist in the Royal Air Force since its inception, that our present aural standards are not too exacting; I will even go further and try to prove to you that in some ways they are not exacting enough. By far the larger part of the chronic middle-ear disease that we encounter in the Service has had its origin before the recruit enters the Service; it is subsequent to the diseases of childhood, to the exanthemata—measles, scarlet fever, diphtheria, etc., that disease of the middle-ear tract commences; over and over again I am asked to examine airmen suffering from middle-ear disease and obtain a history of aural trouble originating before they entered the Service and continuing off and on since; were we to be more rigorous in our recruiting examinations, were the ears of every recruit examined by an expert, I believe that the percentage of sick needing out-patient or hospital treatment—and, more important still, the percentage of invalids—would be reduced to a minimum; this is largely a financial question; every soldier invalided while at home or from abroad is a loss to the State; every day he spends in hospital costs more than his maintenance in barracks, and meanwhile, someone else has to do his work; in 1920 I published in the *Lancet* the results of the examination by aural experts of nearly 50,000 aviation candidates and showed that only 2·8 per cent. of them had been excluded for aural disease; I do not feel that the exclusion of such small percentages should seriously interfere with recruiting but I do feel that the Services, both in matters of finance and physical efficiency, would be the gainers thereby.

My final remarks deal with the attainment of aural efficiency in the peace-time Army. As already indicated I believe there is only one way of obtaining this, and that is by excluding everyone with pre-existing chronic aural disease, or with an upper respiratory tract of the type that experience shows gives a large percentage of aural trouble; we have done this in the Royal Air Force during the last eleven years in the recruitment of aviation candidates; the result has been that the proportion of aural disease among our flying personnel is almost *nil*; every now and again we get, of course, aural disease due to accidents or epidemic diseases like influenza, but the point I want to lay stress upon is that we eliminate the previously damaged ear and the unhealthy upper respiratory tract; in addition, we have obtained an exact description of the nose, throat and ears of everyone at the

time of enlistment, and this must be of great value to the State for reference in cases of claimants for pensions, as also of material assistance to medical officers under the Ministry of Pensions; on parallel lines it should not be beyond the range of possibility to employ only those medical officers on recruiting who have previously had thorough training in nose, throat and ear examinations; I should personally make it obligatory.

Should any relaxation of standards be made when the demand for recruits exceeds the supply, or in the case, say, of another great war? If so, in what direction should this relaxation take place? It is apparent that apart from a number of recruits, who might be temporarily deferred for treatment, the debatable cases would be men with chronic suppurative otitis media, with or without hearing defects who were in other respects perfectly healthy; were it necessary to consider the question of admitting some of these, it seems to me that the presence or absence of symptoms or exacerbations of disease over a preceding lengthy period should play an important part in the adjudication; during the war the Canadian contingent had a system of grading men into categories according to their aural disability, and such a system could be used again in case of a national emergency; it would appear incontrovertible, however, that a man sound and healthy in all respects is more useful and successful over a period of years than one with any physical defects which limit his utility; in civil life much is being done to help us by school examinations and the attachment of aural specialists to hospitals for infectious diseases; let us hope the time will come when owing to these and similar measures, the young adult population will be practically free from aural disease, and it will then be unnecessary for medical officers to examine the ears of recruits; in any case, by progress and co-operation on these lines we will eventually obtain a far higher physical standard among our recruits and reduce to a minimum any inefficiency among our soldiers and sailors due to aural disease.

Mr. Sydney Scott, M.S.

In so far as middle-ear disease is associated with more or less deafness, discharge from the ear, pain, head noises, and giddiness, it is a recognized disability.

The disability may be trifling, interfering but little with the amenities of social life, or it may be harassing enough to interfere with earning power. Some, by reason of deafness, can barely earn enough to be self-supporting, while a certain number are so affected as to be dependent on the rest of the community. The disease may prove fatal, is only temporary, or it persists for years.

It will be our endeavour to ascertain the relative frequency of these various events.

Relative frequency of Blindness, Deafness, and other Disabilities.

Estimates of the value of a normal man to the State have been elaborately considered by the Statistical Department of Industrial Assurance offices, but few companies publish any figures of their business owing to its competitive nature. However, some results of inquiry are issued by the Metropolitan Life Insurance Company of New York. The valuation figures are expressed in dollars per annum. Up to the age of 18 the average normal or disabled boy is an expense, and his value is deferred. Between 18 and 19 he should take a sufficient share in the world's productive work to render himself self-supporting until advanced in age. At least 1 per cent. of the population is seriously handicapped or wholly incapacitated as wage earners by various physical or mental impairments. There are computed to be over 1,000,000 such persons incapacitated by various causes besides ear disease in the United States to-day, and probably about half that number in the United

Kingdom and Irish Free State. Of these persons, deafness so severe as to class them with deaf mutes, there are 45,000 in the United States, 20,000 in the United Kingdom and the Irish Free State.

It is estimated that the average earnings of a normal man in U.S.A. are about $2,000 per annum. Of the blind who can be gainfully employed, their average earnings are only $560 per annum for males, and $300 for females. £115 or £60 per annum respectively, or about $7,000,000 per annum in all, is produced by those able to work. As it costs the United States $37,000,000 per annum to maintain its blind, the result is an actual liability of well over $30,000,000 per annum, while the total loss of services of 75,000 blind represents a potential fall in assets of $150,000,000 per annum.

The economic losses due to deafness are actually and relatively much less. There are only 45,000 so deaf as to be entered as such in Census returns of the United States, compared with 75,000 who are blind. Rather less than half these numbers would apply to our own country. Moreover, 60 per cent. of the deaf males can earn an average of $1,000 per annum, and 18 per cent. of deaf females can earn an average of $500 per annum. As a class they are essentially just self-supporting, but the effect of their disability reduces earning power from a potential asset in man value of $90,000,000 per annum, to about $16,000,000, showing a net potential loss of only $74,000,000 per annum. Thus blind and deaf together cause a potential loss of $224,000,000 per annum, or about one-tenth of the grand total potential loss of all physically and mentally unfit persons in U.S.A.

In computing the burden of those similarly handicapped in the competition for employment in this country before the war, we may take rather less than half the measure of the burden carried by the United States.

Our potential losses would amount to £200,000,000 per annum, of which blindness and deafness account for £20,000,000 and deafness alone for about £7,000,000.

Let us console ourselves by realizing that these are potential, not actual losses; in the case of the blind, the actual burden borne by the physically fit members of the community being only about £3,000,000 per annum, computed on an industrial company's basis.

The deaf are just self-supporting, and, as a whole class, are no burden on their fellows. The number of very deaf persons in the United Kingdom as revealed by each successive census, every decade for fifty years, is stated to be just over 20,000—or one deaf person for every 18,000.

We must note that these are only the hopelessly deaf, including deaf mutes. Many of them are children below 10. From the age of 10 and upwards in England and Wales there are 13,450 recorded in the National Census returns Of these 6,785 are unemployed, while 6,665 are in various occupations.

The following list shows the nature of employment obtained.

	Number of very deaf, employed
In Government offices employed as clerks, messengers, etc.	11
Domestic services	788
Agriculture and fishery	571
Machinery	503
Building	485
Furniture trades	470
Brick-making and pottery	153
Mining and quarrying	151
Printing and paper-making	238
Textiles	407
Dress-making	1,829
Inns and hotels	194
Gas, water, electric supply and sanitary services	22
Various professions and trades	1,843
	6,665

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The commonest of occupations in which deaf persons were employed according to the various census returns are as follows.

Men	Women
Boot-making	Dress-making
Tailoring	Domestic service
Farm labourers	Laundry service
Gardening	Tailoring
Carpentry	Shirt-making
Cabinet making	Charing
Painting and decorating	
French polishing	

Among the more educated classes of deaf persons there are some occupied as painters, sculptors, lithographers, photographers, bookbinders.

Authors, journalists and proof-readers, in banking as ledger clerks. Solicitors—drafting deeds or accountancy.

Architects, draughtsmen and engineers.

It is practically impossible for a very deaf medical student to become qualified, but medical men who have become too deaf to practise can devote themselves to histology, bacteriology, radiography and some special branches of medicine or surgery.

So far we have considered only those who are very deaf in both ears.

We must now consider those with only a limited defect in hearing. This is an extremely common condition, but no figures are available to show the frequency.

It has been stated that only about one-third of the community have perfect hearing in both ears. We know there is an ample margin provided by nature, so that hearing power may be considerably diminished before the loss becomes noticeable.

We sometimes meet those who have lost a considerable part of each ear drum membrane, from middle-ear disease, without being noticeably deaf in ordinary circumstances, though tests reveal a definite defect in hearing power.

To estimate a normal standard of hearing, "the residual whisper" should be heard at 20 ft. at least, with each ear separately.

I asked Mr. Banks-Davis, who has had so much experience in testing candidates for the Civil Services, what rules he adopted before recommending certificates for permanent establishment. I append his reply as a basis for consideration, having regard to the rather exacting requirements of His Majesty's Postmaster-General and of other heads of departments in the Civil Services:—

(1) "Deafness in one ear need not disqualify except in the classes mentioned below, provided the hearing is good in the other ear.

(2) "One tympanic membrane ruptured by accident should not necessarily disqualify.

(3) "Cases in which a radical cure of middle-ear disease by mastoid operation has been effected may be considered six months after operation, for an ordinary certificate.

(4) "Cases with a history of suppurative otitis media if inactive at the time of examination may be given a 'special recommendation' for two years.

"The following cases should be ineligible:—

(5) "Marked deafness[1] in both ears.

(6) "Suppurative otitis media *active* at the time of examination.

(7) "Cases of deafness in which there is reason to believe that the disease is progressive.

[1] Inability to hear the voice whispered at 10 ft. in either ear is considered by Mr. Banks-Davis as ground for rejection.

(8) "Candidates for the post of Officer of Customs and Excise, also of Telephone and Telegraph Services, whose hearing is not normal."

If it is true that only one in three persons have perfect hearing it is fortunate that military requirements did not necessitate perfect hearing in both ears during the late war.

Indeed, we know that some degree of defective hearing, or even the presence of certain forms of middle-ear disease, so long as evidence of the latter is not obvious, is quite compatible with employment in many civil occupations as well as in a military capacity. Nevertheless, for reasons to be given later, it is important that every defect discovered in the ears of an applicant for service in the Army or Navy should be carefully recorded, especially in an otherwise eligible man. It would seem that a sufficient practical guide for a recruiting medical officer so far as the ears were concerned, was to accept an applicant who answered questions immediately when spoken to in an ordinary tone of voice, without showing any evidence of deafness, that is without leaning forward, turning the head or putting a hand to the ear. Experience gained during the course of the war has shown that something more was necessary. A considerable proportion of men had disorder of the middle-ear at the time of enlistment or mobilization, and a large number had had discharge for years.

This did not necessarily prevent many of them being good and useful soldiers; as civilians they were accustomed to the discharge and neglected the ear unless in pain or discomfort. But owing to the injuries resulting in disease of the middle-ear caused by the war, it became difficult to distinguish those cases with disorders which had originated before the war from those arising by reason of the war.

The medical officers acting on the Medical Boards concerned, for the Ministry of Pensions before and after demobilization, must have had what was often an impossible task to separate correctly these two classes. When in doubt, men have received pensions or gratuities to which they must often have had no just claim, from other members of the community.

The following regulations were recommended to be applied to the Canadian Contingent respecting hearing conditions in order to grade men according to hearing or efficiency.

Category A.

(1) Hearing ordinary voice at 15 ft. or better in each ear and no organic disease of the ear.

(2) Hearing 21 ft. in either ear, but little or no hearing in the other ear, with no active organic disease.

Category B.

(1) Hearing 15 ft. in either ear, and little or no hearing in the other, but with no active disease in either ear.

(2) Hearing better than at 12 ft. in each ear, but with discharge from either ear of moderate degree, or subject to frequent recurrent discharge from the ears of a moderate degree.

Category C.

(1) Hearing reduced to less than 15 ft. with freely discharging ear or ears.

Men recently recovered from a mastoid operation would be in this category for three months, and then re-graded if possible.

Category D.

Those with signs of offensive discharge from the ear, free formation of granulations, recurrent pain, if regarded as incurable by an otologist, to be regarded as permanently unfit.

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It will be realized that men in category B and C will require special ear treatment from time to time, and those in category D may become transferable to higher categories after appropriate special treatment.

It is quite practical to make an inspection of the drum membrane of the ear of every man passed fit in other respects for enlistment or mobilization. It would merely be a matter of arranging team work. Large numbers would quickly pass the tests. Others would need expenditure of time to grade those with defects. A precise statement of the hearing and the condition of the middle-ear and drum membrane when abnormal could be recorded. The value of such records would be unquestionable, both during a man's service and on demobilization, when considering those who may be granted pensions or gratuities.

I have obtained the following data which have some bearing on the relative gravity of ear disease.

At the hospital with which I am connected the number of new out-patients with ear disease has gradually increased from 800 to 2,400 per annum in twenty-one years. The majority of these have middle-ear disease. On an average each patient attends four times a year, so that among these cases there are nearly 10,000 attendances a year.

About 10 per cent. of the new patients are admitted as in-patients. Rather more than 4,000 have been admitted for disease of the middle ear. Of the total number 287 have died from complications; the main causes of the fatalities were meningitis, brain abscess, lateral sinus thrombosis. In considering any modification in Army regulations with regard to the hearing of recruits (I do not include the Royal Air Force, as for this branch the conditions would be more rigid) one would presumably exclude the obviously deaf, those with obvious otorrhœa in either ear, also those having visible granulations or cholesteatoma. But if a man is not obviously deaf, if there is a clean perforation in one drum only, especially in the lower half, even if there is an occasional slight discharge from the ear, should he be rejected? I would recommend that all recruits having purulent rhinitis and disease of the upper air passages, with perforated drums or deafness, should be referred to civil hospitals, and recommended to re-apply after treatment.

It may seem paradoxical to uphold the strict rules of the Air Force and yet refer to the successful career of flying officers with bilateral otosclerosis. I knew one well before and after he became a pilot. To make him hear in ordinary circumstances one had to shout. He heard well in a machine and in a noise. Another officer had large perforations of both ears, and was also very successful. These officers never became giddy from rapid diving. In peace time such men would be excluded, yet in war they succeeded well, and so there seems reason to modify the requirements for peace and war time respectively.

Major JOHN HARE.

In view of the urgency of the aural problem at present confronting the Army medical authorities, I will try to explain as concisely as possible what this problem is:—

The annual number of recruits medically examined on enlistment fell to its lowest just before the war—i.e., to 43,000. Immediately after the war it rose to the high figure of 86,000, but since then there has been a rapid and sustained descent to the present low value of 49,000. These figures do not represent the total number of men presenting themselves for enlistment, since many more are eliminated by recruiters and do not arrive at the stage of medical examination.

Given a low rejection rate, the present low figure would usually meet the Army's requirements in recruits. Conversely a high rejection rate makes it extremely difficult to meet requirements.

Since 1921 a very high rejection rate exists, and although falling slowly it is disproportionately high considering the rapid fall in the number of recruits medically examined. One of the chief causes of this high rejection rate is *aural trouble.*

The recruiting problem in short is therefore as follows :—

When it is increasingly difficult to attract recruits, increasing numbers are being rejected and this is partly due to increased stringency in aural examination. The Army Medical Authorities have therefore seriously to consider " whether the present somewhat stringent regulations, bearing on recruitment of men suffering from aural trouble without accompanying deafness, cannot be modified to permit of the acceptance of those unlikely to break down or become deaf during their period of Army and Reserve service."

The following facts seem to show that the present aural standard *is* unnecessarily exacting and therefore is capable of modification :—

(1) Prior to 1914, when there were more men serving with aural disease than after the war and when ear examination was comparatively perfunctory, the rates for admission with ear troubles, for invalids sent home and for invalids finally discharged were insignificant compared with the years 1921-26.

(Reason) If it can be proved that the incidence and severity of otitis media have not greatly increased since the war, it is reasonable to assume that, since so large a number of men are now prevented by ear trouble from enlisting, there must have been a higher proportion of men serving before the war with the condition than at present.

(2) The death-rate due to ear disease or its sequelæ is inconsiderable.

(3) The present standard is impossible, for reasons to be given later.

The present aural standard was decided upon by a special committee in 1925 and is as follows :—

No recruit should be enlisted who suffers from any of the following defects: (1) Deafness ; (2) aural discharge ; (3) perforation ; (4) radical mastoid operation ; (5) dermatitis of the meatus (eczematous or desquamative) ; (6) conditions which after the removal of the cerumen prevent thorough examination of the drum (e.g., atresia of meatus, hyperostosis, and other neoplasms) ; (7) conditions giving a history of recurrent headache, deafness, tinnitus or vertigo which cannot be accounted for by the state of the upper air passages.

The ear examination will consist of : (*a*) The removal, if necessary, of all cerumen. (*b*) Auriscopic scrutiny (difficult cases may be referred to the nearest otologist). (*c*) Hearing testing (must be able to hear strongest whisper at twenty paces).

Owing to the circularization in India of a letter in December, 1924 (the substance of which was afterwards embodied in Regulations A.M.S. India) to the effect that all soldiers suffering from chronic suppurative otitis media must be invalided to England, the numbers so invalided from India rose from 100 in 1924, to 404 in 1925, and of these a large proportion were discharged as permanently unfit. As the return of these men and their replacement resulted in serious financial loss to the State, I was instructed to examine the ears of all recruits in the British Isles with under six months' service. This examination showed that in addition to the large number of men prevented by aural disease from entering the Army, 4·6 per cent. had escaped the vigilance of the medical examiners of recruits and the medical officers in charge of depots. This shows that if the present regulations were given full effect, the number of men prevented from enlisting would be extremely large—probably 10 per cent. One is therefore forced to the conclusion that the present Regulations'

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must be modified if only because their efficient carrying out demands a knowledge of otology which the average medical man does not possess.

As regards the effects of aural disease upon efficiency in the Army, the following important fact was elicited: "Of recruits discovered to be suffering from aural disease or its effects, in less than 10 per cent. was aural deficiency suspected owing to inability to hear orders on parade." Now to discuss the question: "To what extent does aural disease affect efficiency in the Army?" This I will discuss under the headings (1) Home Service; (2) Service abroad; Service in war. At the same time, since a reduction in our present standard must result in a larger percentage of men serving with aural lesions than at present, I will give my views as to measures for dealing with such increases.

Home Service.—The following requirements in training may aggravate or bring about acute exacerbation in existing otitis media (in nine out of every ten men found suffering from aural disease during service the defect was present on enlistment):—

(a) Exposure; (b) bathing parades; (c) boxing; (d) exposure to concussive effects (rifle and machine-gun fire, etc.).

Exacerbations do occur at some time or other in the majority of cases and the symptoms are then so intensified as to compel the man to report sick. Medical officers, thanks to no fault of their own but to inadequate aural training, are unduly apprehensive regarding ears (examination requirements are in terms of complications which rarely ensue), and, since otological specialists are conveniently available at home, are too ready to "pass on the baby." This is deplored chiefly for two reasons: (1) In doing so it proves to the man's satisfaction that he harbours a lesion which his medical officer refuses to deal with and he soon finds his aural disability very useful whenever he desires to avoid uncongenial duties. (2) The specialist otologist, as such, must give the man such treatment as he considers necessary and under present conditions the following procedure is usual: (*a*) Where the aural lesion is mild (40 per cent. of cases) and there is no aggravating lesion in the nose or throat necessitating operative interference, the man is sent back to his unit with instructions as to treatment. (*b*) Since aggravating or causative lesions are found in many cases, about 40 per cent. require admission to hospital for operation. If aural discharge continues despite operation combined with special meatal treatment, the man is sent back to his unit for such meatal treatment as is considered suitable, i.e., the period in hospital is reduced to a minimum.

(*c*) If despite operative interference, a soldier keeps returning to hospital with recurrences, as only a selected few can be given a radical or modified mastoid operation, he is discharged the Service. Owing to the large number of cases of chronic otitis media in the Army, and because a real cure for these chronic aural conditions would need a radical mastoid operation in about 50 per cent. of cases and because it is very often impossible to prejudge the effect of operation upon the hearing (i.e., many would be discharged through loss of hearing resulting from operation) radical mastoid operations are, with few exceptions, performed only as a life-saving measure (20 per cent. discharged as permanently unfit).

At present one ear-nose-and-throat specialist is employed in each Command. Even in existing circumstances he has almost more work than he can cope with. If more men with aural lesions are to be accepted more specialists must be employed. Medical officers must receive aural training to equip them to undertake their legitimate share of the increased responsibility.

Service Abroad.—My remarks apply chiefly to India because it furnishes the highest invaliding rate for aural trouble, and otitis media is more prevalent in India than at home and is more subject to recurrences.

The otitis media usually found is an exacerbation of an otitis media with perforation which existed on enlistment. In my opinion the perforation is the most important factor during service abroad. In stations where swimming baths exist, aspergillus infection of the external auditory meatus is very prevalent—with a perforated drum and exposed tympanum this infection only too often extends via perforation and results in a middle-ear infection. This is often of a mixed type and consequently resistant to pure meatal medication.

Where there are no swimming baths, the infecting agent is probably dust. In either case recurrences are frequent, and because in India aural specialists are few and their visits to stations infrequent—owing to the distances to be travelled—medical officers are compelled to treat such cases, often for lengthy periods. In such circumstances it is natural to expect that many cases are brought up for invaliding.

If by relaxing the present standard still more men with aural trouble are to be sent to India, it is essential: (1) To employ more aural specialists in India; (2) that medical officers receive such aural training as will enable them to undertake the added responsibility and to discriminate between lesions trivial or important.

War.—It is common experience that Thomas Atkins is more likely to report sick on trivial grounds during peace time with its monotonous round of duties than during the sustained and everchanging excitement of war. This being so, aural trouble is not so manifest in war time, and wastage from this cause is comparatively unimportant. Nevertheless it was reported that during the War, owing to the unwarranted fear of complications on the part of medical officers ignorant of ear diseases, innumerable mild ear cases were sent to the base, which could perfectly well have continued full duty at the front.

To minimize such wastage as does occur and which will increase if a more lax aural standard is adopted, the solution remains the same as under the two previous headings: (1) Provision of an inadequate number of aural specialists; (2) training of all medical officers in otology.

Conclusions.—(1) Although efficiency is only slightly affected by aural disease, the present inadequate aural knowledge among the medical profession as a whole results in a loss to the State, and until this is rectified, it would be best to exclude all men suffering from aural lesions.

(2) If the present economic position will not allow of this and such men must be accepted, otology must occupy an important part in the training of R.A.M.C. officers and more specialists must be employed.

(3) When this is achieved the present standard could be reduced to the following, capable of execution by all medical officers of recruits: No recruit should be enlisted who suffers from deafness or from obvious aural discharge.

Surgeon Commander Maxwell.

The effects of aural disease on the efficiency of the Navy can be discussed under four headings.

(1) *Loss of Working Time.*—There were 14,637 days' sickness due to aural disease in 1925, a figure only exceeded by such large headings as, "stomach," "intestine," "catarrh," "venereal disease," and "injuries," = £10,000 approximately. Four-fifths of this lost time was spent in hospital. The figures are magnified by the fact that a man must be fit for any service in any climate before discharge, and may be sent to a small ship quite isolated from specialist assistance. A ship may be at times more isolated than any land force.

(2) *Deafness Impairing Efficiency.*—Good hearing has increased in importance with the increased complexity of ship life, and the universal use on board of telephones, hydrophones, and the increasing importance of communications.

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(3) *Invaliding.*—156 men were invalided in 1925 for aural disease, out of 1,412 from all causes, a number only exceeded in diseases of eye and tuberculosis. The figure represents a considerable loss in time, training, transport home in many cases and professional attention.

Death.—Only three deaths occurred in 1925, out of 254 from all causes, and except as a potential catastrophe, the loss under this heading is small, but the possibility affects the disposal of aural cases. How real it is can be seen by its effect on the terms of any insurance company.

Types of Aural Disease met with.—(1) Suppurative otitis media. This is an overwhelming majority of all cases, and nearly all the wastage comes under this heading. We may divide the cases into: (*a*) Recrudescent, which are merely relapses from disease which occurred before entry. (*b*) Acute cases in young ratings, who in many cases have predisposing defects in nose and throat. (*c*) Chronic otitis media, in older men. Many of these are incurable except by radical operation, and are best invalided *sine die*. The presence of polypi should be an absolute cause for invaliding.

(2) " Dry " deafness, from sclerotic conditions and Eustachian obstruction; here again, the long treatment needed and the doubtful prognosis make invaliding the most efficient line of action.

(3) Nerve deafness.—This is, in my experience, uncommon, and when internal ear deafness is found, it is more often due to syphilis than to gun-fire. Permanent gun-deafness is not common, and though temporary dull hearing after heavy firing occurs, it is not a serious cause of inefficiency. Traumatic rupture of the drum is also rare, though cases may be seen only when infection has occurred.

(4) Otitis externa.—In the tropics especially this is a common and at times difficult condition for the non-specialist officer to diagnose and treat without hospital assistance and moral support.

This problem of the losses of time and money due to ear disease in the Navy is best met, in my opinion, by:—

(1) The specialist examination of recruits:—No rules as to what lesions are admissible and what are not can replace the judgment of one trained to estimate the value and future of a patient's ear by special examination. It is only too common to find that a recruit has been accepted during what is merely an interval in his disease, or with an aural lesion bound to light up under unfavourable conditions. Present Army standard seems not too exacting.

(2) The rejection, or at least early treatment, again by specialists, of defects in the nose and throat likely to cause aural breakdown. Conditions in a modern warship impose great strain on the upper air passages, and together with trying climatic conditions make the removal of gross defects essential.

(3) The maintenance as far as possible of specialist officers at sea for service as consultants to fleets or squadrons, particularly on foreign stations, to whom other medical officers may refer for advice in the disposal and treatment of ear cases.

(4) The increased attention to dental treatment, which is now the rule in the Navy, will undoubtedly help to lessen all septic conditions in ear, nose and throat.

(5) The quicker and earlier decision to invalid men whose lesions are certain to persist, always remembering that more time and trouble must be devoted to save a fully trained man, who is anxious to remain in the Navy, than a young man on whom no money and training have been spent, or who may be anxious to leave or at least indifferent.

(6) The accurate keeping of records giving the condition of the drums and tuning-fork reactions, is of great importance, and renders the diagnosis and assessment of disabilities much more sound and thorough.

(7) Malingering is not very common, but here again the services of a specialist are essential, for a determined attempt to feign deafness can only be bowled out by one skilled in the use of the tuning-fork, and at home with all the tests that may trip up the false statement.

Otorrhœa and ear disease cannot be ignored in the Services, for they are uncleanly, and depressing when not dangerous, and are often if not always only part of a syndrome affecting nose and throat as well, and reducing general health and morale.

LIEUTENANT-COLONEL T. B. LAYTON

said that his experience of the subject was very different from that of Wing-Commander Ranken, yet after preparing his notes for these remarks he had found, on reading the advanced proof of Commander Ranken's paper, that his conclusions were practically identical with those of that officer. All he would do, therefore, would be to pick out some of Commander Ranken's comments, and perhaps vary the emphasis laid upon certain of them.

The first point on which he would lay stress was the vast difference between the subject when considering peace-time service and service during such a national emergency as that through which we had been recently. For members of the Services probably the peace service possessed the greater interest, while to those who had returned to civil life, and hoped never to have to rejoin the Forces, the aspect of the national emergency had the greater attraction.

Taking then the three symptoms of deafness, giddiness and discharge, he thought that in peace-time all these should exclude a man from entry into the Services. Of the three he would have regarded poor hearing as of the least importance, and would have been prepared to admit a man who had moderate deafness. Wing-Commander Ranken had, however, produced reasons for excluding such a person, and it was for those in the Services to decide to what extent deafness was an impairment for a man's work in them.

With regard to giddiness, the important point was the possibility of malingering. This he would divide into subconscious malingering—which was scarcely distinguishable from hysteria in many cases—and true malingering, in which the man was trying to "swing the lead." It was, however, very difficult to prove either type of malingering in a given case and, therefore, it was almost impossible to insist upon a man going back to duty if once he had complained of giddiness.[1]

Turning to discharge from the ear: the main question was the possibility of any individual developing meningitis and dying from it. In the majority of cases this would not happen, yet might not the particular man whom one was examining be the one out of many in whose case meningitis would ultimately develop? If, then, a man in an outlying station reported sick in consequence of it, the medical officer must send him down to a hospital where operative treatment would be possible, if necessary. And if the man were sent immediately back by the ear specialist and reported sick again in a week's time, the medical officer would have again to send him down. If anyone asked why he did this, the answer would be that he might have developed meningitis, and it was not fair to keep away from surgical aid a man with symptoms where this possibility might arise.

On active service during a national emergency, discharge was not, of itself, an excuse for going down the line. The risk from meningitis was not great and, compared with the danger that a man went through in a field unit, it was practically negligible. In the Egyptian Expeditionary Force during the Great War, an order

1 *Vide* "Malingering and Allied Conditions of Deafness," *Journ. Laryng. and Otol.*, 1923, xxxviii, 146.

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was issued that no man was to be sent down the line merely on account of ear discharge, and he thought that this order practically stopped the journeying back of these patients to the base.

Did this argument, however, apply to a smaller war, such as the South African War of 1900, which was not a national emergency in which every man had to do something? A man with a running ear might develop acute mastoid disease or meningitis when he got overseas. Was it fair to subject him to that risk in addition to the others which he voluntarily undertook when he joined up? In his (the speaker's) opinion it was not. When then the medical officer made the examination previous to a unit going overseas on active service, all these men would have to be left behind. For these reasons he would say that active suppurative otitis media should exclude a man from being admitted to the Service under ordinary circumstances in peace time. He would lay stress on the difference between active chronic suppurative otitis media and residual chronic suppurative otitis media. By the latter he meant those cases where there was evidence that at some time there had been suppuration in the ear, but that there was none now and had been none for a considerable period past. He did not see why these cases should not be taken just as were persons with a normal drumhead. There was no reason to suppose that they were more liable to acute infections than a person who had never had a damaged ear. A certain number of each category would get an acute infection of the upper respiratory tract and develop acute disease of the mastoid requiring operation. Nor did he see why perforation *per se* should prevent a man being admitted into the Forces. If the perforation had a healed margin and the middle ear, as seen through the perforation, appeared to be dry, he considered it safe to take that man.

With regard to the doing of radical or modified radical mastoid operations, he would advise officers in the Services to leave these operations alone. The man who had had a radical operation performed on him was as liable on active service conditions to break down and have a discharging ear as he was with his suppurative otitis media before he had the operation. He (the speaker) had had considerable experience of this at the Ministry of Pensions, where for six years after the War he had been treating men who had had radical mastoid operations performed. In many of these cases the cavities though previously dry, had broken down and suppurated afresh the moment the conditions under which the men lived became other than hygienic. The thin, poorly-nourished epithelium ulcerated; this was followed by a discharge and the whole train of symptoms started afresh. His advice was that if a man needed a radical or a modified radical operation he had better be invalided out of the Service.

He did not lay so much stress as some did on the loss caused by illness of partially-trained soldiers. He did not think it could be a bad thing, from a national standpoint, to have a large number of partially-trained soldiers in civil life. As a pacifist he hoped that there would for many years to come be a steady reduction of strength in the actual number of the fighting Forces, but as a practical man he had to admit the possibility of these men being used again, and he thought the permeation through the nation of men who had had some training was a distinct advantage. This furnished an answer to Wing-Commander Ranken's question as to what was to be done when the demand for recruits exceeded the supply. He would advise that the various units had better remain below establishment until the number of men applying to join the Forces rose again, or until the establishments were reduced to the number which was coming forward.

His last point was to insist upon the importance of an otological examination of every recruit. This was so important that it ought to be done—firstly, that there might be an efficient record of all men while they were in the Service, but also to save the money of the nation after such men left the Service. He thought there

must be a great number of men who had no note of pre-existing disease entered on their enrolment form, and who had to have their disability included as attributable to war service, whereas it was more than likely that their ears were already damaged before they joined up. He believed that such examination was possible. Modern medical education was changing. Nowadays every student had to work in the ear department before he took his final examination, and every medical officer in the Services in the future should be able to make an otological examination of reasonable efficiency in the majority of cases. In those in which they were doubtful the men might be referred to the specialist. He agreed with Major Hare that more specialists were needed in the Army, and he thought that the increased cost of such officers would be more than compensated by the economies to be effected by proper otological examination.

Mr. T. Ritchie Rodger.

I will put forward for consideration a point about which confusion appears to exist, viz., the relationship between middle-ear suppuration and internal-ear deafness due to excessive noise. The gun-deafness which afflicts naval gunners and artillerymen belongs to the type of internal-ear deafness variously classified as noise-deafness or occupation-deafness. In some text-books on otology one meets with the statement that occupation-deafness is predisposed to by middle-ear disease. Hence examiners of recruits for certain branches of the Services may be inclined to reject men with any middle-ear defect at all, on the assumption that they are more vulnerable to the effects of gun-fire, and medical officers in charge of men who become the subjects of middle-ear diseases may be similarly influenced.

So far from middle-ear disease conducing to gun-deafness, the very opposite is true; most middle-ear conditions are a protection against noise-deafness.

The pathological condition in gun-deafness is an atrophic degeneration of the nerve-endings in the cochlea, due to trauma from the impact of excessive sound waves, and, as I shall show, the injurious sound waves reach the cochlea by way of the tympanum, not by bone conduction. It should, therefore, be expected that anything that would reduce or damp the normal conducting power of the tympanic membrane and the chain of ossicles, would tend to lessen the injury to the delicate nerve-endings of the cochlea. In middle-ear disease we always have, in greater or less degree, such interference with the normal conductivity. That middle-ear disease is a protection against noise-deafness has been proved both experimentally and clinically.

I am not aware of any post-mortem investigation of the microscopic condition of the cochlea in subjects of gun-deafness, but we may safely assume that it is the same as that found in animals which have been subjected to excessive noises and detonations. I shall make brief reference to the work done on animals, but anyone wishing to go into the subject might look up a paper I contributed to the *Journal of Otology* in March, 1915.

Wittmaack, of Jena, in 1907, published findings from the examination of the cochlea of guinea-pigs killed after exposure to loud sounds. In Corti's organ the changes ranged, according to the severity of the pathological process, from slight swellings of the hair-cells and their supporting cells, to so extensive an atrophy of the whole end-organ that nothing of it remained beyond the merest fringe of flattened epithelium. Cases of moderate severity showed vacuole formation in the hair-cells and rod-cells; the latter were no longer upright, and the tunnel-space was consequently contracted. Dieter's and Hensen's cells had lost their characteristic structure, and in their place was a heap of cubical and cylindrical cells becoming gradually more and more flattened.

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In the nerve-fibres in the spiral lamina very marked changes were also seen. Individual fibres had lost their uniform calibre, showing alternate swelling and narrowing. Actual segmentation was seen in the more severe cases, and in most cases proliferation of interstitial cells had occurred. In the ganglion cells of the spiral lamina disappearance of Nissl's granules, vacuole-formation, the presence of bodies like "asthma-crystals," changes in the nucleus, which had taken on an irregular contour and become homogeneous in structure. In the more severe cases the vacuoles were very large and the protoplasm shrunken.

In his first series, Wittmaack used an electric bell suspended in the animals' cage, and found no change in the cochlea. The changes described were observed only after connecting the bell to a metal floor on which the animals rested. He therefore concluded that the injurious sounds were bone-conducted. In the following year, however, Yoshii, Von Eicken and Hoessli, all working separately, found identical cochlear changes in circumstances in which bone-conduction was discounted.

It was realized that this question of bone- *versus* air-conduction was of the highest importance from the point of view of the application of the investigations, i.e., the prophylaxis of noise-deafness in certain occupations, including gun-firing and boiler-making, and each observer concentrated on this. Yoshii, besides demonstrating the cochlear changes without using a metal floor, proved his contention in two other ways. One of his animals developed middle-ear suppuration, and it was found that the cochlea on that side had escaped the pathological process, while that of the other ear presented the expected changes. With another animal he used a revolver shot at close range (20 cm.). The right ear showed a large perforation of the drumhead with bleeding in the middle ear—no change in the cochlea. The left ear with intact drumhead and middle-ear normal, showed cochlear changes similar to those described.

Von Eicken took a series of animals in which he had removed the incus on one side. In every case he found that the cochlea on that side had escaped injury, while the other internal ear showed the typical pathological changes. Hoessli confirmed this experience and also Yoshii's experience with middle-ear suppuration. He also made experiments with a rubber mat to eliminate bone-conduction and still found the cochlear changes.

Habermann, of Prague, in 1906, published the post-mortem findings in five subjects of boiler-makers' deafness. The changes in the cochlea were similar to those described, but one patient had had a unilateral middle-ear suppuration, and on that side the cochlea was normal.

In 1914 I made a clinical investigation of a series of boiler-makers. It was found that every man examined, even including boys who had been only a short time at work, showed deafness for tuning-forks corresponding in pitch to the predominant sounds of the boiler-shops, while all who had been a considerable time in employment had deafness extending more or less over the whole tone-scale. This deafness was invariably of the nerve variety. Of ninety-six ears examined, however, six presented signs of active middle-ear disease, and all of these failed to conform to the signs of nerve-deafness. Five others had a history of suppuration of short duration in earlier years, but presented healed drumheads, and these ears suffered from nerve-deafness. The obvious conclusion from this clinical investigation as well as from the experimental work, is that a middle-ear condition capable of interfering sufficiently with the ossicular conduction of sound waves is a protection against noise-deafness.

From these considerations two points arise:—

(1) The prevention of noise-deafness in naval gunners and artillerymen is to be achieved by the regular use of protective blocking of the external auditory canal.

Rubber mats for the gunners to stand on, as has been advocated, are useless. Plugs of vaselined wool, jeweller's wax, celluloid or rubber may be used, and their use should be made obligatory.

(2) A recruit with a slight middle-ear defect in itself not of a nature to exclude him, should not be rejected on the assumption that since his hearing is defective he is likely to be vulnerable to the effects of gun-fire. Anything that interferes in any way with the normal conduction apparatus is by so much a safeguard to the cochlea, and a protection against noise-deafness.

Sir James Dundas-Grant

said that, with regard to the difference in the danger between a perforation in the central or near the anterior part of the membrane, with a thick purulent discharge, and one in the postero-superior segment, close to the attic and aditus, the first was comparatively harmless, while the second was dangerous owing to the tendency for the epithelium lining the meatus to proliferate into the antrum and mastoid cells, and for the formation of cholesteatoma. In the less dangerous variety, if there was room for microbes to get in, there was also room along the same route for the escape of their products. The otologist at the front should draw this distinction and realize that a man with cholesteatoma was in more danger than one who had a perforation lower down.

Mr. Ritchie Rodger had made out a strong case that adhesive disease in the tympanum was a protection against gun-deafness, but the experience of Moure, of Bordeaux, in a large number of cases, was that men with nasal, naso-pharyngeal and throat trouble were usually more liable to suffer from concussion-deafness owing to gun-fire than others. The physics of it was different; it was known that when the full force was not expended upon an entire membrane there was no concussion. A result of his (the speaker's) experiments was that when the membrane gave way the internal ear often escaped.

There should be a routine otological examination in recruiting; he hoped that the authorities would see the great necessity of appointing a larger number of otologists in the Services, unless the Army was to be much reduced. Army medical officers, more perhaps than other practitioners, should be well acquainted with the diagnosis and treatment of ear conditions.

Colonel P. H. Henderson

said he regretted that he could not agree with Mr. Layton's view that if we could not get the numbers required for the fighting services, acting on the standards recommended by the otologists, the Services should be cut down accordingly.

Such a policy, with the dental and other specialists also pressing for perfection, would soon so reduce the fighting Services that they could not fulfil their proper functions. The strength of the Services was for the Cabinet to decide, on the advice of the Army Council, and the physical standards adopted, while aiming at the highest possible physical efficiency, must provide the numbers required from the material available. It was chiefly a matter of supply and demand, and he would remind the meeting of the quality of the raw material available.

In 1918 the National Services Medical Board found that of every nine men of military age in Great Britain, on the average, three were fit, two were infirm, three could almost be described as physical wrecks, and the last was a chronic invalid with a precarious hold upon life.

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To-day, the percentage of physically fit amongst volunteers for enlistment was approximately forty, and of these, twenty-two were cast for other reasons—under age, bad character, etc.—leaving only 18 per cent. fit in all respects for the Army, under present standards.

The 1922 Report of the Chief Medical Officer of the Board of Education showed that 35 to 40 per cent. of children of school age suffered from physical defects.

He (Colonel Henderson) was afraid that, while all desired to see the fighting Services composed solely of A1 men, that laudable desire could not yet be fulfilled. It was possible, even probable, that mother and child welfare schemes and the greater care now taken of school children might go far to help, perhaps in twenty years, but the eugenic child was not yet the rule—rather the exception.

The Army was short of recruits, and to get the desired numbers it might be necessary to lower certain physical standards; the problem was, which standards could be lowered with least effect on efficiency?

For example, should the standard of height be lowered, while maintaining a high standard in regard to special disabilities? A man of 5 ft. 2 in. of normal physical development could not carry the infantryman's present kit and fight effectively. Moreover, short men could not keep up with taller men on the march; and nothing makes a worse unit than a mixture of high- and low-grade categories.

On the other hand, every Service officer has seen and treated as out-patients cases of various ear diseases with few, if any, untoward results.

Major Hare had shown that in pre-war days mastoid operations, and deaths in the Service due to ear disease, were rare. It was common knowledge that the 1914 British Expeditionary Force contained many men with ear troubles such as now caused the rejection or discharge of a recruit and the invaliding of a trained soldier, yet it was the finest fighting force produced by any country in the Great War.

These facts led one to consider the policy prevailing in India until recently, of sending home all men suffering from "perforation of the tympanum" or "suppurative" or "ordinary" discharge from the middle ear.

The Annual Health Reports of the Army for 1923 to 1926 showed, amongst those invalided, only twelve cases of perforation of the tympanum, but no fewer than 1,297 cases of suppurative, and 404 cases of ordinary, inflammation of the middle ear.

The numbers invalided yearly for these conditions from 1923 to 1926 were between 400 and 500, and as the large majority came from India, the cost to the State must be something like £80,000 a year. There was nothing in the Reports to show whether the men suffered from such deafness or loss of physical powers as to render them unfit for military duties.

He advocated the extension of specialism in the Services, provided that specialists applied their expert technical knowledge to prevention and treatment, and did not let it obscure or distort their vision in such questions as recruiting and invaliding. He suggested that if the policy, particularly in regard to invaliding, were changed, much money now wasted could be saved to the State, with a fraction of which sufficient extra otologists could be provided to ensure, by efficient treatment, the retention in the Army of men now being invalided.

This policy would relieve non-specialist officers of anxiety in regard to the risk of meningitis or other complications in middle-ear cases. He himself had treated many men and retained them with their units without untoward results or personal anxiety, although they were suffering from various forms of ear disease.

In conclusion, he asked the following questions:—

(1) Was there any record of how many of the ear diseases were the sequelæ of influenza?

(2) Was he right in suggesting that, as a rule, post-influenzal cases yielded comparatively easily to treatment, and that in those cases the ear trouble did not, as a rule, tend to recur, although it might do so as the result of a subsequent attack of influenza?

(3) Had any attempt been made in India during the past few years to ascertain the cause of the ear disease in the various cases, and to cure or alleviate before deciding to invalid?

(4) Was there any record of the treatments adopted for the various ear troubles, and of their duration, cost and results?

(5) As the men proceeding to India were all found fit for general service before embarking, had their conditions become so aggravated since arrival in India as to render them unfit for military duties?

(6) In what way had their ear troubles made them physically unfit, many of them within a few weeks of their arrival in India?

Wing-Commander J. Macdonald

said it seemed that the Royal Air Force had not yet struck the recruiting difficulty which appeared to have been a source of trouble to the Army. The Air Force figures had not varied much during the last few years as to the proportion of rejections for aural defects.

He agreed with Wing-Commander Ranken that there seemed no reason for altering the Royal Air Force standard of admission; on the present standard it was not likely to be difficult to get the men required. Possibly in an emergency it might be wise to modify the standard.

The Air Force was perhaps fortunate in that the system of examination was more or less centralized in London. In 50 per cent. of the candidates there was a preliminary examination by a local doctor, and if the man was considered fit, he was sent to the London Recruiting Depôt, where, if there was doubt as to the candidate's aural condition, experts were easy of access.

Mr. Alex. R. Tweedie

agreed with Mr. Layton as to the need of separating the subject into two categories: peace and war. In the Egyptian Expeditionary Force, towards the end of the Great War, there were many men serving with chronic suppurative otitis media, and many of these took part in the final successful attack at Gaza.

The routine at that time was to send such cases for examination by a specialist, who, if he considered them suitable, returned them to the regimental medical officer, with a note as to the diagnosis and treatment. Under such circumstances he considered this constituted a most appropriate procedure.

With regard to peace-time recruiting, he thought that, if possible, every man should be excluded who had intranasal, postnasal or aural lesions or until such were rectified. In the Nottingham General Hospital it had been considered advisable for all the nurse-candidates to pass the otologist's scrutiny of nose, naso-pharynx and middle ear. He was very glad to hear Mr. Sydney Scott stress the importance of rhinitis. Surgeon Commander Maxwell had urged the same thing—and he himself would emphasize the prime importance of a healthy nose and naso-pharynx in consideration of all inflammatory aural lesions.

He could not agree with Mr. Ritchie Rodger as to the protective possibilities of a pre-existing middle-ear lesion. Whatever the results of experimental investigation, a very large percentage of internal ear lesions, in pensioners examined by him since the War, showed evidence of pre-existing tubal or middle-ear affections.

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MR. LESLIE POWELL

said he agreed with Mr. Ritchie Rodger about suppurative otitis media, but not as to catarrhal otitis media without perforation. During the War he had examined many men who suffered from gun-deafness. In those with retracted drums and no perforation, deafness was particularly marked and persisted.

All who alluded to pensions in this discussion took the national point of view. He considered it very hard on an individual if he had passed the tests and served in the Forces, and then, on the plea that he had pre-existing disease, was refused full pension, if he was found after the War to have been rendered inefficient for his civil occupation, whereas the point was that deafness would have been negligible in these cases if the effects of gun-fire had not been added.

Sections of Odontology, Electro-Therapeutics, and Pathology.

[*March* 26, 1928.]

DISCUSSION ON THE PULPLESS TOOTH.

Mr. Frank Coleman.

Abstract.—A large proportion of the population have pulpless teeth, hence the importance of this subject. The main principles concerned in the treatment of pulpless teeth are: the removal of the pulp in its entirety, the drainage of the stump of the severed pulp, the cleansing and drying of the root canals and the filling of the tooth cavity.

Local anæsthesia is preferable to caustics for desensitizing the tooth pulp prior to its removal. Antiseptics used in a dry condition are preferable to those used in a moist condition for root canal treatment.

Nothing is gained by filling the root canals, if the peri-apical tissues have scarred there is no further discharge into these canals; and if they have not scarred the filling of the root canals will only obstruct drainage. If the discharge from the canals is copious, the tooth cavity should be left open to the mouth except for the insertion of a loosely placed dressing.

Untreated pulpless teeth are more liable to give rise to "open" sepsis; treated pulpless teeth to "closed" sepsis.

The crux of the question as to whether pulpless teeth should be retained or extracted is one of evaluating their advantages and disadvantages.

A tooth deprived of its pulp owing to *infective* changes gives rise to subjective and objective symptoms and signs.

A tooth deprived of its pulp owing to *physiological* changes gives rise to no apparent ill effects.

The importance of this subject will be realized when it is disclosed that more than half of the population have pulpless teeth in some form or other, and that in ten per cent. of the population the number of pulpless teeth may be from five upwards. I assume the term "pulpless tooth" to denote a tooth from which the pulp has been removed and which has been subsequently treated by filling, crowning or some other means. I am excluding teeth and roots with dead pulps that have been untreated, although I shall refer to these for purposes of comparison.

A pulpless tooth should not be designated as a "dead" tooth, for the periphery of the tooth (i.e., the cementum) retains some vitality through its periodontal membrane. It would be just as incorrect to designate as a dead tooth a pyorrhœic tooth, stripped or partly stripped of its periodontal membrane and consequently deprived of some of its nutrition. That there is a separate blood-supply—and therefore, probably a separate nerve-supply—for the pulp of the tooth and for its periodontal membrane is evidenced clinically in our everyday work. When we anæsthetize the pulp of a tooth by applying a local anæsthetic directly to it, we find that the periphery of the tooth (i.e., periodontal membrane) is not rendered anæsthetic, and, whereas we can remove the pulp painlessly we cannot extract the tooth without pain: conversely, we can remove a tooth without pain by injecting a local anæsthetic around it, yet the pulp of that tooth will retain some degree of sensibility. In both cases there is a slight blunting of the sensation, peripheral or central, according to whether the drug to the tooth is applied centrally or peripherally. This slight loss of sensation to the adjoining tissue in either case can be accounted for by the anastomosis that takes place between the pulp and periodontal vessels.

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The main principles concerned in the treatment of teeth with exposed live pulps, or teeth containing dead pulps, are:—

(1) To remove, as far as possible, every trace of pulp tissue. In teeth with live pulps the desensitization of the pulp must first be assured. This can be brought about by the employment of a local anæsthetic or of a caustic (usually arsenic); if a caustic be employed, a few days must elapse to bring about the complete devitalization of the tooth-pulp. The method by local anæsthesia is more scientific and will leave the tooth in a less unhealthy condition.

(2) To cleanse the root-canal or canals of the tooth, and to treat the peri-apical tissues via these channels.

(3) To fill the cavity of the tooth when the peri-apical tissues have ceased to discharge through the canal of the tooth and have formed a scar at the tooth apex.

Most dental surgeons recommend filling the roots of the tooth as well as the cavity in the crown of the tooth; personally, I only fill the cavity in the crown of the tooth (i.e., pulp chamber), as by this means the peri-apical tissues can be further treated if and when necessary; the empty root canal serving as a passive channel down to these tissues.

If the root canal be occluded with a filling, further treatment directed to the peri-apical tissues by way of the tooth becomes impossible, and extraction of the tooth is often the only means left of draining these tissues; whereas, if the canal be left empty, drainage can be provided for in the event of the tooth (or more correctly its surrounding tissues) subsequently becoming inflamed and painful.

The method of treatment of pulpless teeth that I have adopted for nearly a quarter of a century, depends upon the supposition that a scar forms around the apex of the tooth soon after the removal of the pulp, whether the pulp be removed after desensitization by the aid of a local anæsthetic or by the aid of a caustic. This method of treatment, I may claim, is on a more secure basis than a purely hypothetical one, for we know that if an arsenical paste be applied to the skin a slough is formed, and on the separation of this slough a scar remains. We have no reason to suppose that the arsenic acts differently in the case of a tooth pulp. Clinical observation supports the view that the process is the same for a few days after the removal of the tooth-pulp; provided it be not virulently infected, dressings placed in the tooth-canal remain clean and dry, showing that no further discharge takes place into the root-canal. Virulently infected teeth may require very many dressings before this condition is reached, if it is ever reached, and it may be necessary in the earlier dressings to leave the tooth cavity open in order to provide better drainage.

Some adverse comments on this method of treatment were brought forward in an editorial in one of the dental journals in 1906, but they did not shake my convictions. The editor quoted from two authors their reasons for the filling of root-canals. The first author quoted gave this reason:—

(1) "The object of root-canal filling is the maintenance of an aseptic condition in a sterilized root-canal by hermetically closing it at both the apical foramen and the orifice, and thus preventing its infection by the entrance of either fluid or gases."

The second author quoted gave this reason:—

(2) "The main object in filling the pulp canal is to hermetically close or seal the apical foramen, and thus prevent the egress of septic matter, mephitic gases, micro-organisms, or their waste products, from the canal into the apical space, and also to preclude the possibility of the entrance by transudation of fluids from the apical tissues into the canal."

It is certainly aiming high to fill a root which you cannot see (especially in the days before X-rays were introduced), and in such a way that you hermetically seal the apex or apices against the entrance or exit of micro-organisms. This editor,

however, is a thorough optimist, for later on he tells us in the same editorial, that if the canals of the tooth are small and tortuous, "straightening by enlargement is often so useful an aid to solid filling." His conception of a pulpless tooth is rather curious, for he puts the cart before the horse in the following statement, when he advocates filling the apex of a tooth, and then leaving the tooth on trial before completing its restoration. Most of us would regard the trial stage at an end when once the tooth apex had been filled, especially if "hermetically" sealed. Since the writing of my original paper in 1906, the anatomy of the root canals of teeth has been found more complicated than we realized, for instead of a single unbranched canal for each root, there may be as many as twenty lateral branches, each containing a filament of the pulp passing to the periodontal membrane, and frequently several foramina at the apex of the tooth for the entrance of the pulp.

It makes little difference with what material the crown portion of the tooth is restored, provided the substitute is durable, presents a smooth and unirritating surface to the mouth, and is everywhere flush with its abutment, so that whether the root carries an artificial crown (provided this is not a collar crown) or a filling is immaterial, the implanted portion, abutment, or root of the tooth being the main source of infection. The method, however, by which the implanted portion or root of the tooth is treated prior to its restoration may be of some considerable importance in regard to its after history. Needless to say the technique should be carried out under as aseptic conditions as possible. The employment of a local anæsthetic in preference to a caustic for desensitizing the tooth-pulp prior to its removal has already been mentioned. The use of efficient but not irritating antiseptics (if such exist), and the employment of these in the dry or solid form (e.g., iodoform) rather than in the moist or liquid form, is also important. Finally, the tooth cavity should only be filled when the peri-apical tissues have scarred and, therefore, ceased to discharge. To close the cavity, or still more the canal, of a tooth before the peri-apical tissues have ceased to discharge, or in other words until inflammation has subsided, is to court disaster.

Tomes in his "Dental Surgery," 1897, mentions paraffin as a root-filling and speaks of excellent results attending its employment. He goes on to say, "On opening up such teeth after the lapse of months or years, the pulp-canals were sometimes found to be sweet but empty, the semi-solid paraffin having apparently been soaked up by the dentine, and it has therefore been abandoned in favour of wax with a higher melting point." The fact of the pulp-canals being found "sweet but empty" would to my mind commend paraffin as a root-filling.

Recently evidence has accumulated indicating that pulpless teeth cause less disturbance to their surroundings when their roots are not filled to their extremities than when more completely filled. Grove (*Dental Cosmos*, 1921) found on examination of a thousand radiograms of treated pulpless teeth that there was a smaller percentage of rarefied areas around teeth with partially filled roots than around teeth with roots more completely filled. Bulleid[1] regards the granuloma at the end of the root of a dead tooth as a defensive mechanism on the part of the patient's tissues, and states that this defensive mechanism is more often associated with a partially filled root than with a more completely filled root. The reason why so many untreated dead teeth remain quiescent is that their infected canals lie open to the mouth and can, therefore, discharge into the buccal cavity any inflammatory exudation or gases arising from their putrescent pulps which would otherwise be pent up in the tissues, and sooner or later give rise to abscesses. We find even with the greatest attention to asepsis and technique that we not infrequently cause a "flare up" at the outset of treating these dormant dead teeth, due probably to some

[1] *Proceedings, Roy. Soc. Med.*, 1928, xxi (Sect. Odont.), 21.

infected material from the tooth-pulp gaining access to the peri-apical tissues through the canal of the tooth, or to the admission of oxygen of the air to the organisms already present in the pulp.

As a broad statement we must admit that a pulpless tooth is a pathological entity and as such must be an irritant to the tissues, and the crux of the question whether it should be retained in the mouth or extracted is one of assessing its advantages and disadvantages. The dental surgeon is perhaps best able to assess the disadvantages arising from the loss of a tooth or teeth, as these disadvantages are not only immediate but also mediate, for he has to look to the future as well as to the present of the patient's dentition, whether artificial or natural. The physician is best able to assess the advantages that pertain to a more healthy or less unhealthy mouth.

I have purposely omitted to give the usual list of sequels that may arise as the result of infected teeth; they are well known and depend mainly upon their anatomical relations to the vascular and lymphatic systems, the food and air passages, and to the mentality of the patient.

If the tooth be an important one for mastication, for personal appearance or for the retention of a denture, a reasonable attempt may be made towards its preservation, but this does not imply that the principles of surgery are to be disregarded. These circumstances must be looked upon as misfortunes rather than as factors in influencing the method of treatment to adopt.

The pulp of a sound tooth may die as the result of injury (i.e. trauma). Here we have the condition of a tooth with a dead pulp excluded from outside infection. A large number of these teeth eventually give rise to abscesses, they are almost invariably like all so-called dead teeth, slightly tender on percussion. The abscess may open on to a mucous or skin surface and subsequently continue to discharge through the sinus formed. If the sinus closes or is inefficient for carrying away the discharge a recrudescence of the swelling takes place. In fact most dead teeth require an exit for their discharges, which is, without doubt, preferable to direct absorption of these discharges by the surrounding tissues. Nature's safety valve for dead teeth is usually on the gum near the apex of the tooth or between the tooth and its socket, the discharge in the latter case appearing at the side of the tooth. The least harmful form of an untreated dead tooth is probably that with a sinus or safety valve on the gum or at the side of the tooth and a pulp chamber open to the mouth, for under these conditions most of the inflammatory exudation is swallowed and probably largely destroyed by the intestinal juices. The least harmful form of a treated dead tooth is that with a relatively clean root-canal (whether filled or otherwise) and the presence of scar tissue close around the tooth apex or a peri-apical space capable of self drainage.

Black has shown that molar teeth upon which a patient could bring to bear in closure a pressure of over two hundred pounds, could withstand a pressure of only a hundred pounds or less after devitalization of the pulp. Personally I have never had any experience of a dead tooth or of a pulpless tooth in my own mouth. Clinically we find that these teeth are almost invariably a little tender on percussion as compared with normal teeth. They are less translucent than normal teeth and often present a slight degree of gingivitis at the gum margin and a blush of redness over the tooth-bearing area (especially on the outer aspect). That the tissues will tolerate teeth without pulps is evidenced at the extremes of life. The root of a deciduous tooth may be absorbed to such an extent as to render the tooth pulpless, yet it may be retained in this condition for many years without any apparent ill effects. True, its retention is governed to some extent by the presence or absence of its successor. In elderly people the tooth pulp may undergo concentric calcification until finally the whole pulp-chamber and the root canal become filled

with a mass of calcified tissue, yet these teeth cause no apparent ill effects on their surroundings. This seems to favour the view that the function of the pulp is largely, if not entirely, concerned in the calcification of the tooth, and some physiologists believe that the function of the pulp has ceased when the tooth is fully formed. No doubt it will be argued that these changes at the extremes of life are physiological and not pathological; however, they may serve to throw light on the problem in view. The methods, therefore, by which nature renders a tooth pulpless are by absorption of the pulp tissue in early life and by its solidification in later life. If we could imitate the natural process of pulp calcification that occurs in elderly people by stimulating the pulp to calcify before it had become infected we should no doubt obtain very satisfactory results with our pulpless teeth. Chronic irritation (e.g. erosion, attrition) will sometimes lead to a deposit of protective dentine (secondary dentine) over the pulp and perhaps some day we may be able to promote and control this process. If a tooth with a calcified pulp is capable of maintaining its vitality, a pulpless tooth should likewise be capable of doing the same. The only difference in their nutrition may be the presence of a small bare area deprived of periodontal membrane around the apical foramen of the pulpless tooth.

The treatment to pursue for pulpless teeth is evident in nineteen cases out of twenty, but there is a small residue, probably less than 5 per cent. of cases, which show no gross changes or even changes of any kind either clinically or radiologically in which the treatment to pursue may be doubtful. I do not think the treatment of these doubtful cases will be solved until we have further advanced our pathological and radiological knowledge of pulpless teeth. It will not be solved by any method, or elaboration, of technique.

The operation of excising the end of a root involved in peri-apical destruction does not appeal to me as a rational method of treatment. The extent of disease of the root is incapable of diagnosis, as no line of demarcation is formed between the living and dead portions of the tooth. It therefore becomes merely an empirical removal of a certain amount of necrotic root. The opening and scraping of sinuses connected with dead teeth, the injection of drugs through these sinuses and other such like methods of treatment are only palliative whilst the cause remains.

There is no analogy elsewhere in the body to the condition of a pulpless tooth, for there is no tissue having properties similar to that of a tooth pulp. Sterile metallic substances are tolerated by the body even when they destroy tissues such as the periosteum of a bone (e.g. screws and metal plates used in the treatment of fractures). I have rarely regretted removing a pulpless tooth even when there has been little or no evidence that it is acting as a foreign body, for in most cases I have found one or more of the roots to be discoloured or even necrotic, bathed in a slimy discharge and usually foul smelling. The extraction of a firmly implanted pulpless tooth the root canals of which have been enlarged to receive the metal abutments of a crown, may be far from an easy operation. Yet unless this tooth is removed completely, a source of infection persists as great as, if not greater than, the original source.

Mr. Owen C. Morphy.

I have heard it said that there is no pathology of dead teeth—that they are bad dentistry. I will admit the last part of this sentence at once—in the same sense that all surgery is bad medicine, but otherwise the statement is not consistent. If pulpless teeth are so heinous, there must be a very obvious and conclusive pathology in connexion with them, and I conceive it to be the bounden duty of the pathologists to convince us of their view-point. Mr. Coleman has pointed out the vast number of people with pulpless teeth, and one would like to inquire what percentage of this number carry their pulpless teeth with impunity. The responsibility undertaken with

a septic tooth is obviously greater than where a pulp is devitalized and removed under aseptic or antiseptic precautions. The fundamental conditions underlying such an attempt are that the patient must show evidence of resistance to infection by the possession of good general health, and should not be beyond middle age except in rare instances. The justification for the attempt is chiefly where the saving of such a tooth will obviate the need for a partial denture. I believe most dental surgeons will agree that the partial denture, especially when made of a material such as vulcanite, is a prolific source of oral sepsis, mainly because it so seldom receives the really scrupulous care necessary to make it comparatively innocuous. I believe that a partial denture may very easily lead to a greater degree of infection than arises from one or two pulpless teeth replaced thereby. Admittedly, with the denture the infection is of the open type as compared with the closed apical type.

Unfortunately, it is not, as yet, feasible for the ordinary dental practitioner to check his root-canal treatment by bacteriological tests for sterility, and we are compelled to rely upon clinical signs, which are extremely inadequate, and on radiological evidence for the results of our treatment. A notable change has lately occurred in the interpretation of the radio-lucent area at the apex of a tooth. Formerly it was regarded as the measure of the infection present, and a tooth showing such definite area was promptly condemned to extraction. The apical granuloma is now coming to be regarded as the expression of the patient's resistance to the disease, and I am tempted to suggest that we are in that stage of transition which might be called the stage of the "laudable granuloma." It is the tooth showing no defensive granuloma which is the most dangerous. If, however, the apical granuloma only forms in the presence of drainage, its defensive value must be small, as when drainage is established there is least need for it. Weston Price as the result chiefly of experiments in which he implanted infected teeth under the skin of rabbits, has produced a mass of evidence mainly condemning pulpless teeth. The rabbits nearly all died after periods varying from a few days to a few weeks, having developed secondary lesions in their synovial membranes, hearts, kidneys, etc.

Two obvious criticisms arise—

1. Rabbits are very susceptible to streptococcal infection.

2. They received a sudden massive dose as the result of the operation, giving them little chance of developing immunity.

This is not the manner in which infection occurs in the mouth, except in cases of acute abscess which generally receive drastic treatment. The process is essentially a slow one. When the same experiments were performed on rats with a high natural resistance to streptococci, the teeth were almost invariably either encysted or extruded through the skin, and the animal suffered no damage. Encapsulation with dense fibrous tissue is here the method of defence, and this I believe occurs in many cases of apicectomy. Clyde Davis in the *Dental Cosmos* for 1920 gave a histological analysis of 100 root-filled teeth, in which the root-filling stopped short of the apex. He found in all cases where the canals were not infected and caustic drugs had not been used, that cementum had grown into the apical foramina and completely occluded them. In cases where infection had occurred, cementum had grown into the foramina, but the occlusion was not perfect. Carl Grove, *Dental Cosmos*, 1921, produced similar evidence, which goes to emphasize the need for avoiding instrumentation at the apex so as not to injure the vital cells in the periapical region, and is an argument in favour of Mr. Coleman's practice of not filling canals, which renders over-filling impossible. It further shows that even where infection has existed, the ability of the cementoblasts to lay down fresh material has not been destroyed.

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Dr. H. M. Worth.

The part which radiography plays in relation to the pulpless tooth may be considered under two main headings, namely: (1) As an aid to the diagnosis; (2) as an aid to the treatment.

Radiography as an Aid to the Diagnosis of the Pulpless Tooth.

In these days, when patients are referred to the radiologist for the purpose of finding infected "foci" in relation with the teeth, and when it is considered by many observers that all "dead" teeth are a menace to the health, it is important that every fragment of evidence that will help in the diagnosis of such teeth should be recognized.

There are no intrinsic radiographic changes to be seen in a tooth without a pulp, but there are often certain appearances in or around such a tooth which are of great value in aiding its recognition.

Certain of these changes are absolute evidence that the tooth is pulpless, while others are only suggestive, and it is possible therefore to formulate two categories: (*a*) changes which are certain evidence that the tooth is pulpless; (*b*) changes which are only suggestive.

Certain Evidences that a Tooth is Pulpless.

(1) The presence of filling material in the root-canal or pulp chamber is of course certain evidence that the tooth is a dead one, and as nearly all filling materials contain zinc oxide or some other metallic salt which is opaque to X-rays, its presence is usually quite obvious in a radiograph.

It is the practice of many dental surgeons not to fill the root-canals, but the pulp chamber usually contains some filling whereby such teeth are recognized.

(2) The presence of osteitis with bone destruction, or bone rarefaction only at the apex of a tooth which is associated with loss of continuity in the lamina dura, is nearly always evidence that the tooth is dead, but there are rare occasions when this does not appear to be correct, and when such a tooth gives the reactions normal to a vital condition.

A condition of osteitis at the apex of a tooth often spreads to the adjacent tooth, and its involvement is sometimes recognized by the loss of the lamina dura at the side and apex of the root.

(3) The presence of a large cavity extending into the pulp chamber, with no other evidence is sufficient to warrant the opinion that the pulp is dead. Care is necessary to ascertain that the pulp is actually involved and that the shadows are not superimposed.

(4) The presence of a filling involving the pulp chamber is equally diagnostic.

(5) The pulp chamber and root-canals occasionally become completely calcified, a condition easily recognized in a radiograph.

Changes Suggestive that a Tooth is Pulpless.

(1) Cases occur where there is rarefaction of the bone at the apex of a tooth, and where it is not possible to be certain that the lamina dura is broken in its continuity—such an appearance is very suggestive that the tooth is dead and further tests should be carried out to ascertain the condition.

(2) The periodontal membrane is occasionally seen to be thickened at the apex of a tooth only, which suggests strongly that the tooth is dead, but when this appearance is associated with a large cavity, or filling, or with the presence of slight sclerosis of the peri-apical bone the condition is even more suggestive.

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It must be pointed out at this stage that there is an appearance seen at the apex of the upper canine, which appears to simulate a thickened periodontal membrane, and which might be mistaken for the condition just described, but is apparently normal.

Further, it is sometimes seen that the shadow of the lower molar roots are superimposed over that of the inferior dental canal, which gives an erroneous appearance that the membranes are thickened at the apices only.

(3) The presence of a cavity near the pulp chamber suggests that the tooth is not vital, but care is necessary to determine that the appearance is not due to the superimposition of a buccal or lingual cavity over the pulp.

(4) The presence of a fracture of the root of a tooth nearly always indicates that it is dead, more especially if there is slight rarefaction of the bone at the apex.

(5) The presence of a dental cyst which is seen to be definitely involving a tooth other than the causative one, usually means that such tooth is dead.

The lamina dura must be seen to be broken in order to feel certain that the tooth is involved.

(6) The presence of a "pin" very near the pulp is evidence sufficient to suggest that the vitality of the tooth should be tested.

RADIOGRAPHY AS AN AID TO THE TREATMENT OF A PULPLESS TOOTH.

(1) A radiograph is of use at the commencement of treatment to eliminate the presence of apical infection, and later to follow the effect of treatment on apical osteitis.

(2) It is important to know whether the apex of a tooth under treatment is completed.

A tooth with a completed apex shows the pulp-canal to be converging towards the apex, whereas when the apex has yet to form the canal is seen to diverge towards the apex and the apical foramen to be "funnel-shaped."

(3) To ascertain the number, direction and length of the root-canals, it is advisable to insert smooth metallic broaches before taking a radiograph.

(4) During the process of treatment of root canals it is sometimes necessary to ascertain the presence of pulp nodules in the canal, or of a piece of broken instrument.

(5) Perforation of the apex or side of a tooth is usually seen only in a radiograph when a metallic broach or piece of wire has been inserted into it before radiography.

(6) It is often considered necessary in the treatment of a root canal that the apex should be reached by the filling material, and consequently a radiograph is indicated to ascertain this point. Occasionally the filling will be seen to have passed beyond the apex.

In view of the fact that so much can be learnt concerning the pulpless tooth from a radiograph, it might be thought an easy matter to recognize such a tooth always, but it must be pointed out that such is not the case, for there are many dead teeth which present not the slightest evidence of their condition radiographically, and it becomes necessary for the dental surgeon to use other methods of investigation.

This means, then, that radiography is only a "positive" test of the non-vitality of a tooth.

Dr. G. MURRAY LEVICK.

I propose to keep to that part of our subject to which electro-therapeutics must be confined and which is the chief concern of the physician, namely, the septic sequelæ. Though some of my colleagues may have something to say on the subject of ionization, I believe that light will be found the most valuable physical means of treatment.

Following on the success of solar and artificial light in the treatment of septic infections, attempts have been made to treat oral sepsis by irradiation. I think, however, that the application of artificial light in such cases has hitherto been based upon a misconception. The destruction of micro-organisms by the direct action of ultra-violet rays is a side issue and a quite unimportant factor in such treatment. This is, I think, where the mistake has been made. The clearing up of septic infection in various parts of the body, following a well-directed application of general light treatment, is an indirect effect, due to the conversion of the rays into chemical energy in the tissues, so that substances are synthetized and are distributed throughout the body. The effect on tissue nutrition is often immediate and marked, and it has been shown that the bactericidal power of the body is raised. The formation of vitamin D and consequent stimulation of the calcium metabolism is one example which has found explanation and proof in the laboratory. No doubt there are other processes following the application of light. In the case of ultra-violet radiation, we know that the skin is the tissue concerned in the conversion of the rays. Therefore in seeking to produce vitamin D in the body by radiation, the skin is the selected tissue, even though we wish to influence the nutrition of the skeleton. We are, moreover, seeking natural channels in giving such treatment. When we direct ultra-violet rays upon the mucous membrane of the mouth the reverse is the case.

The evolution of mucous membrane has been carried out under conditions which protect it from ultra-violet rays, therefore it would be absurd to turn to the mucous membrane in an attempt to produce immunity indirectly in place of the skin. Surely it is also absurd to attempt the destruction of bacteria in infected gums and teeth by the direct application of ultra-violet rays, whose lethal effect is so superficial. Moreover, any appreciable destruction of bacteria must in this case be accompanied by a corresponding destruction of mucous membrane and is a form of irritation that has always appeared to me to be most unwise.

The treatment of oral sepsis by general ultra-violet radiation of the skin is sound in every way and indicated just as strongly as in cases of sepsis elsewhere, on account of the secondary and residual infection of distant tissues. But I strongly deprecate the application of ultra-violet radiation to mucous membrane and think we should view with special alarm the practice of its application to the throat, which has already been followed by terrible results. Following the valuable lesson we have learnt in the application of ultra-violet rays to the skin and the certainty that it is a perfectly natural process, we should approach the direct treatment of oral sepsis by light from a similar standpoint. In seeking to correct pathogenic conditions it is a sound rule first to study the conditions under which evolution has produced and preserved the particular tissue to be treated, and to use the natural agencies disclosed in each case, rather than the more unnatural methods too often employed as a result of our early medical teaching. One of the facts disclosed in the course of this study has been the preference given, through evolution, to the red rays in the measures adopted for protecting tissues from solar radiation. The ultra-violet rays are absorbed and utilized by the skin and rarely pass beyond it. The violet, blue and green rays rarely pass beyond the superficial fascia, excepting special situations. Yellow rays pass the fascia where this is not very thick and penetrate fat for some distance, though they are absorbed by it fairly rapidly, but they do not enter the closed mouth. These facts I have ascertained by spectroscopic experiment and, in some cases, on tissues within a minute of their removal on the operating table. When we reach a wave length of 600 mm. however (that it is to say red light) we find that the deeper tissues are permitted to receive and absorb it to such an extent that in bright sunshine most of our joints that are not covered with clothing must be quite brilliantly illuminated with red light. When the skin is pigmented as a result of solar radiation

the red rays are allowed to pass through the pigmented skin, whereas the others are arrested. An experiment familiar to those who search for inflammation of the antrum is the placing of an electric light in the mouth of the patient in a darkened chamber. Red light, and red light alone, issues from the front of the face as it would pass through the fabric of a Japanese lantern. Therefore the rays have traversed, in some situations (e.g., through the antrum), mucous membrane, with its abundant blood-vessels, two layers of bone, facial muscles, skin and fascia. It was a revelation suddenly to realize that in bright sunshine the mouth and nasal accessory sinuses are lighted with red rays, while the others are excluded. It was this revelation, together with the other observations above recorded, that led me to treat with success a variety of pathogenic affections of these regions, including acute and chronic inflammation of the antrum. Spectroscopic examination of the rays from a lamp after they had passed through the tissues in front of the mouth showed that they were composed, as far as the visible rays were concerned, of absolutely pure red light, and I will show it is unlikely that the infra-red rays penetrate very deeply beyond the skin, so that we can exclude them also in treating the mouth. One of the factors concerned is the absorption of the rays by water, and I will now throw on the screen figures taken from the Smithsonian physical tables which show clearly that the absorption of rays by water becomes suddenly very marked beyond the red region, so that these heat rays must be absorbed by the water in the tissues before it reaches any considerable depth. Blood provides a water screen with a red pigment and would thus practically cut out all the rays of the spectrum excepting red. Thus as far as sunlight is concerned we may say that almost all the deep tissues receive and absorb only red light and if any direct physiological effect is produced in them by light it must be by the red rays alone. We have little scientific evidence as to the way in which red rays may act in the production of immunity, excepting that phagocytosis becomes increasingly active at a temperature somewhat above that of the blood under normal conditions. It seems possible that metabolism of cells other than the phagocytes may be similarly affected. I have made further reference to this subject in a paper read before the International Congress of Radiology in London in June, 1925, and published in the *British Journal of Radiology*, May, 1926. Owing to the penetration of the red rays through mucous membrane and bone, care should be taken not to direct them upwards through the region of the soft palate. In some cases a more less or severe headache may follow a few hours after such an application, due, I believe, to the rise in temperature at the base of the skull. We should be very careful, therefore, in applying red rays to the region of the throat. To sum up, I suggest that, as far as light is concerned, the treatment of chronic oral sepsis should be approached in two ways, direct and indirect. Directly through the agency of the red rays, indirectly through the general radiation of the skin by the rays of the natural or artificially produced solar spectrum, as indicated by the treatment already common to many other conditions. In dealing with secondary and residual infection following aural sepsis this is a wise measure and I can say from experience that it is attended with good results.

The treatment of mucous membrane by ultra-violet radiation is dangerous and unjustifiable.

Dr. MEYRICK-JONES

said that in pulpless teeth pain was not generally present. If they were not infected at first, they had a predisposition to infection. In general, when the radiograph showed pathological changes—or often even when it did not—the teeth were extracted. If they were not, periodical radiographs should be taken. In the treatment of cases referred to the dental surgeon there should be close co-operation between himself and the physician.

Dr. A. Livingston

said that he joined issue with Dr. Worth in his effort to diagnose the vitality of a tooth. If members examined any dry skulls to which they had access, taking out the teeth from the sockets, they would find multiple foramina in the bone below every apex; the lamina dura was not continuous for 2 mm. from the bottom of the socket. It was known that a cyst, half an inch in diameter, might show involvement by its walls of teeth adjacent to that which was causing the cyst. Those teeth were stripped of some of their periodontal membrane, yet they were frequently alive. He had cut sections of teeth where there were granulomata and abscesses and cysts, yet the teeth were "alive" and showed nerve fibres in the apical part of the pulp.

In discussing the pulpless tooth, one must remember the possibility of the pathogenicity of the rarefied area so frequently—as radiology disclosed—associated with it; small masses of granulation or scar tissue would be found at the apices of root-filled teeth when they were extracted; but still the actual value of the associated pathogenicity was difficult to evaluate.

The question of apicectomy was an important one. For this to be a success it must be premised that the case on which it was performed was a chosen one, and that the treatment was carried out with as good a technique as the operator could master. The younger generation was exercising the utmost caution in treating a tooth—whether one which must be rendered non-vital or one which was septic when it came to the dental surgeon's notice—because it was not known what was beyond the apex or might reach that region.

Dr. J. F. Brailsford.

A tooth which has been deprived of its pulp has lost its vital power, and inasmuch as it contains within its unfilled nerve-canals and dentinal tubules (estimated as measuring three miles in length), dead organic matter which is brought into association with the circulating blood through the dentino-cemental foraminæ, it is liable, even if sterile when the pulp was extirpated, to be infected by any organism circulating in the blood.

It has been shown that even when caries has only extended one-fourth of the way through the dentine towards the pulp, that approximately 50 per cent. of the pulps of such teeth are infected and further that it is impossible even with the most scrupulous care and the use of the best known bactericidal medicaments, to be certain that the infection has been eliminated. It is probably rare for the nerve canals to be completely filled, even though the canal may be simple and straight, as most of the substances used cannot be made accurately to fill the canal; and, owing to absorption of the solvents used in the preparation of the filling medium, this contracts and leaves a space, which with the dentinal tubules is unprotected by any vital agents and liable to become the storage place for bacteria and toxins. When the nerve-canal is delta-like or has lateral openings, the pulp remnants cannot be removed nor can the canals be suitably filled even with impregnation methods.

Bacteriological examination has shown that, no matter what medicament is used in the treatment of infected apices, any dressings left in for 48 hours are found to be infected, and that, even when all care has been used, root-filled teeth may be a source of great danger to the patient. Experimentally it has been proved that such root-filled teeth may contain bacteria or toxins which, together or separately, produce toxæmia and death when inserted into animals.

Mayo says: "Although the possessor of a tooth with dead pulp without local reaction may be enabled to eat better for a time he is conducting his health on borrowed capital as he may not have a physician or dentist who will appreciate that the disease or broken health that

may develop is due to such an apparently trivial cause, that persons comparatively well may later develop a variety of diseases that destroy health and happiness if not life."

While Inglis says: "At present it looks as though no root-filled tooth can be considered other than as a life risk while sterile filled roots can be infected from other sources of infection of which there are many."

The Radiographic Appearances of Pulpless Teeth.

It must be clearly understood that the evidence which the radiograph gives is only a "skeleton" picture of the pathological lesion. The actual lesion, certainly in bone pathology, is always of much greater extent than the radiograph suggests to an untrained observer. Therefore in dental conditions where the important changes are relatively small in size it is important to have radiographs which will show these details. Fortunately, the close proximity of the radiographic film to the tooth enables a sharp detailed picture to be obtained which will permit of magnification, and as the danger of the lesion is not to be judged by its size such magnification will not mislead by showing the slight changes to be definite irregularities. The outline on the radiograph of a normal tooth enlarged to the size of the radiograph of the femur head is seen to be quite as regular as the latter, whereas the outlines of teeth which show "slight" changes are markedly irregular—a femur-head so irregular would be judged to be gravely involved.

Root Fillings.

The radiograph will show any filling which is composed of opaque material and give some idea of the relative sizes of the filling material and the nerve-canal. It will not, as a rule, show more than the central nerve-canal. It has been suggested that the radiograph will give an indication of the direction of the nerve-canal in teeth with curved, irregularly disposed roots and thus give the dental surgeon the necessary direction for the complete cleansing and filling of the root-canal.

While the radiograph may be useful in a few cases for this purpose, most radiologists who have had experience in radiographic localization will agree that in most cases little help can so be given.

Radiographs, particularly if stereoscopic, of an opaque wire in the nerve-canal will show whether the wire extends to the apex of the tooth or beyond, or whether it perforates the root.

Radiographs of root-filled teeth show that often much shrinkage of the filling preparation occurs, as in fig. 1:—

Price says: "It is to be remembered that a small portion of a gangrenous pulp beneath a root-canal filling is equivalent to an entire gangrenous pulp as a cause of pericementitis. The vast majority of cases occur as a sequel to putrefaction of the pulp, either before or after instrumentation or as a result of infection of the apical tissue by instruments either unsterilized or re-infected by contact with oral fluid, septic fingers, etc."

Septic Condition of Pulpless Teeth.

Weston A. Price, who, with a team of dental surgeons, bacteriologists and laboratory workers, has carried out a very extensive investigation in dental sepsis, considers that 10 per cent. of teeth with septic roots give no indication of this on the radiograph but even so, most of the process illustrations which he used to illustrate the "failure" of the radiographs show definite though "slight" changes, though such illustrations cannot give the detail of the original radiographs.

There is no doubt in my mind that this figure of 10 per cent. can be considerably reduced by skill in the radiographic technic and the care exercised in examining and interpreting the radiographs.

Further, I am of the opinion that *every tooth which is producing systemic disturbance shows definite changes on the radiograph.* The importance of this is apparent when we remember that the clinical tests, even when skilfully employed, may fail to reveal quite extensive lesions.

Burchard and Inglis state that: "In cases of granuloma or blind chronic apical abscess there may be no subjective or local symptoms of disturbance cognizable to patient or operator."

This patient (fig. 5) was suffering from a toxæmia and was sent for an X-ray examination of her teeth. She was annoyed at the suggestion that her illness was due to her teeth and informed me that she paid regular visits to her dental surgeon, who had recently carefully examined the teeth and reported them to be quite sound.

Price, who has made an exhaustive study of the individual and family clinical histories, clinical, radiographic and bacteriological examination of the teeth of 681 selected cases in a series of 1,400, has classified patients into three groups. He says that dental infections tend to produce the same type of tissue reaction around the teeth of different members of the same family.

Group A.

The radiographs of the teeth of this group of patients show very extensive rarefaction around all the involved teeth, and often extensive pyorrhœa (as in fig. 6).

Price regards this local bone absorption as a sign of a good reaction and resistance to the infection on the part of the patients.

Such patients, he says, rarely show symptoms of the rheumatic or degenerative disorders and accordingly classes them as having "absent susceptibility."

Nevertheless such patients come to hospital complaining of gastro-intestinal or other disorders probably brought about by the septic teeth.

Clinically the gums show discharging sinuses and the teeth are often loose, readily anæsthetized and easily extracted; the sockets heal with great rapidity and without discomfort or secondary infection.

Group B.

In this group of patients the radiographs of the teeth show similar changes to those in Group A, but the peri-apical areas of rarefaction are bounded by a zone of sclerosed and therefore denser bone (as in fig. 7).

Price regards this sclerosed bone as a sign of breaking down of the local resistance to the dental sepsis with the entrance of the septic material into the blood-stream.

Clinically there are signs of old fistulæ and a history of former tenderness. Anæsthesia in these patients is less easily produced in this group than Group A. The teeth frequently are difficult to extract and the sockets do not heal so readily.

He says that these patients have acquired a susceptibility to the dental infection and that while no rheumatic symptoms were seen when the condition was acute such symptoms have begun to appear and they clear with the removal of the septic teeth.

Group C.

The radiographs of the teeth of this group show condensing osteitis around the infected root with little rarefaction (as in figs. 8 and 9).

This appearance Price regards as a sign of lack of local resistance to the dental sepsis.

Clinically there is no evidence of fistulæ—the teeth are seldom tender. They appear to be more liable to caries but usually free from pyorrhœa. Anæsthesia of such teeth is very difficult and they are often very difficult to extract. There is great tardiness in healing and the sockets tend to become infected and painful. He says hat these patients have an inherited susceptibility to the rheumatic group of disease.

This classification tends to explain why some patients with extreme and obvious dental sepsis have often few of the symptoms usually associated with septic absorption while others with marked rheumatic symptoms have often no obvious dental sepsis and the radiographs of their teeth show relatively slight changes—so slight that when indicated by the physician or radiologist the dental surgeon is loath to extract the teeth.

Most physicians will agree that these dental groupings are correct with regard to the susceptibility to rheumatic infections, but few I think will agree to the ideas on local resistance.

An examination of the radiographs showing bone sepsis of different types permit of the classification into three groups, which resemble those indicated by Price.

Group A.—Patients with bone sepsis due to pyogenic bacteria in which the radiographs show rapid absorption or erosion of the infected bone. In these cases there is often a large amount of pus found.

As the condition of the patient improves and his resistance to the infection is increased, the rarefied bone increases in density and the patient shows little or no sign of rheumatic disorders even through the degree of septic absorption as judged from the patients condition was very severe.

Group B.—Patients who formerly showed radiographic changes similar to those in Group *A*, but owing to the separation of sequestra, or the inclusion of some septic foreign body, healing is very slow and the radiograph shows that the surrounding bone is sclerosed and very dense. This sclerosis most of us look upon as of the nature of a protective reaction against a chronic infection and not as a breakdown in the resistance of the patient, though such patients often do gradually develop rheumatic symptoms due probably to the long period of slow septic absorption.

Group C.—Patients suffering from non-specific infective arthritis. The radiographs of the infected joints do not suggest ordinary bone sepsis; rarefaction, erosion and sclerosis may be seen, but the changes are slow to form. They resemble the changes in the teeth of Group *C*. Numerous bacteriological researches have been made to attempt to discover the causal organism; various organisms have been suggested, but the lack of uniformity in the findings suggests that either the condition is produced by different organisms, or by some bacteria or toxin which has not been discovered.

That the removal of one tooth with only a small area of rarefaction may be followed by recovery from a profound systematic disorder was shown by Dr. Leonard Mackay in the paper which he wrote in 1922, in which he described the important clinical symptoms which are associated with hidden dental sepsis.

Price has shown that teeth from patients in this group even when boiled retain some toxin which is fatal to experimental animals.

He also found that extracts from such teeth passed through a Berkefeld filter are also toxic to animals.

One of the most important points that he has brought out is that the septic teeth from which absorption is taking place in patients with an inherited susceptibility to the rheumatic and degenerative disorders, produce very little in the way of local clinical signs, and radiographic signs which are small in magnitude.

Radiographs with less marked changes are frequently seen as in fig. 10, which shows a large area of rarefaction due to acute infection chiefly to the lateral side of the root-filled central incisor. The root filling is not very opaque. It will be noted that the sclerosed pericemental bone is "wiped" out about half way to the apex on this lateral aspect and to a lesser extent on the medial aspect of the lateral incisor.

In some cases infection of the peri-apical area leads to the development of large cysts, which, in the upper jaw, may be mistaken for extension of the antrum, as in fig. 12.

Concomitant with the peri-apical erosion, hypercementosis may occur and the roots assume a bulbous appearance on the radiograph. Such teeth may be very difficult to extract. In one case recently where the radiographs showed similar changes to fig. 13, the dental surgeon had great difficulty in extracting the teeth, and he told the patient that there had been a mistake in the radiographic report, as the tooth was too firmly held to have been diseased.

Brubaker reports that neuralgia, functional blindness, and deafness, chorea, epileptiform fits, paralysis, cardiac neuralgia, insanity and other related conditions have been cured by the extraction of hypercementosed teeth.

In contradistinction to hypercementosis we have erosion of the apices as a result of sepsis as in fig. 14.

When the infected apex is near the antrum the infection may pass into the antrum, as in fig. 16.

Radiographic Pitfalls.

The errors in dental radiographic diagnosis may be due to : (*a*) Faulty photographic and radiographic technique ; (*b*) misinterpretation of the radiograph.

With regard to (*a*), it can be said that the manufacturers have done their best to produce an X-ray unit which is as simple in working as any snapshot camera. It is common knowledge that with either kind of apparatus the proportion of films of good quality is dependent upon the care, skill and experience of the operator.

The simplicity of the apparatus will enable the most ignorant beginner to obtain chance successes, and these are stored and exhibited and give a sense of self-satisfaction which tends to prevent further progress.

It is not to be wondered at, therefore, that marks on radiographic films due to faulty technique may (*a*) be interpreted as pathological changes—which are not found at subsequent operation ; (*b*) mask a pathological lesion which is present.

I have known of a number of cases in which the pathological lesion was missed because the films were flat. No amount of clinical or pathological knowledge will enable anyone to give a diagnosis from a faulty film.

Misinterpretation of the Radiograph.

This is due chiefly to : (1) Lack of experience in interpreting radiographic shadows ; (2) lack of knowledge of general and dental pathology.

With regard to (1) every radiologist who has correlated the radiographic and clinical, operative or post-mortem findings, knows that the radiograph may show only slight changes, even when a massive lesion is present. Thus, with acute inflammatory lesions of the skeletal tissues, the radiograph may give no sign ; even with the enormous development which one sees in some cases of periosteal sarcoma ; the radiograph may show nothing abnormal or perhaps a little localized ossification of the periosteum.

One can almost say, particularly in acute conditions, that the more attractive the physical signs the less the radiographic signs. In the case of dental radiography, as the lesions are often small, it is all the more important to pay attention to the finer details. (2) There is no question that for the best interpretation of radiographs the observer must have a good knowledge of the normal and pathological anatomy of the part.

Most books dealing with the radiography of the teeth instance the interpretation of the mental and palatine foraminæ and loculi of the maxillary antrum as areas of peri-apical absorption, so that these mistakes are hardly likely to be repeated except by the beginner; even if they are, the worst that can happen is the sacrifice of one or perhaps two sound teeth.

The result of the wide knowledge of the faulty interpretation of these foraminæ has no doubt been responsible for the much more grave error which is brought to one's notice from time to time, i.e., the interpretation of a definite peri-apical absorption as a normal foramina or sinus. It would almost seem that the larger the cyst, if in the maxilla, the greater the possibility of it being interpreted as a shadow of the antrum.

This is of great importance, because the systemic disease from which the patient is suffering continues or increases in severity, as the cause has been overlooked and allowed to remain, there being no local signs apparent to the patient or clinical observer.

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FIG. 1.—Radiograph of root-filled lateral incisor and canine, showing retraction and shrinkage of root filling preparation. The nerve-canals are shown to be much wider in calibre. There is also an area of rarefaction of the peri-apical bone of both teeth and erosion of the apex of the lateral incisor.

FIG. 2.—Radiograph showing root filling in bicuspid roots. The filling projects beyond the apex in one root and an area of rarefaction has been produced in the surrounding bone.

FIG. 3.—Radiograph showing root filling in bicuspid roots with areas of apical erosion and absorption. A small fragment of an instrument had been broken at the apex of a root and is lying in the rarefied area.

FIG. 4.—Radiograph of root-filled upper molar teeth, showing retraction and shrinking of preparation in anterior root and erosion and absorption of the alveolus around the distal root which shows calcareous deposits. The floor of the pulp chamber had been perforated and a deposit of calcareous material is shown on the projecting root filling.

FIG. 5.—Radiograph showing large "cyst" (probably arising from the root-filled bicuspid) which has eroded the apices of the first bicuspid, canine and central and lateral incisors.

FIG. 6.—Radiograph showing extensive alveolar and peri-apical absorption.

FIG. 7.—Radiograph showing a large apical abscess with sclerosis of its bony wall and erosion of the tooth apex. The lateral incisor is root filled and there is a little erosion of its apex.

FIG. 8.—Radiograph showing small areas of rarefaction at the apices of the bicuspid and first molar with sclerosis of the surrounding bone. (Patient suffering from pains in the back and along the course of the sciatic nerve as well as in the shoulder joints.)

FIG. 9.—Radiograph of molar with one root filled with opaque material the other showing no sign of a nerve canal. There is sclerosis of the pericemental bone and secondary caries beneath the amalgam filling. Patient suffering from neuritis—condition improved on removal of tooth.

FIG. 10.—The area of rarefaction may not always be at the apex of the tooth as many teeth have lateral canals through which the pericementum may be infected.

FIG. 11.—Radiograph showing root-filled lateral incisor and canine. On the bicuspid side of the canine the pericementum will be seen pushed away from the side of the middle of the root. There is marked erosion of the apices of both teeth and very little root filling which shows signs of shrinking and retraction.

FIG. 12.—Radiograph showing a large cyst with erosion of the apices of the central and lateral incisors which have had the pulps extracted and the root-canals partly filled.

FIG. 13.—Radiograph showing "bulbous" roots of root-filled molar with peri-apical absorption.

FIG. 14.—Radiograph showing large area of peri-apical absorption with erosion of the apices of the first bicuspid which shows secondary caries beneath the filling. There is a similar but less marked change in the second bicuspid.

FIG 15.—Radiograph showing sclerosis of the pericementum due to the large filling and a normal antrum.

FIG. 16.—Radiograph showing erosion of the wall of the antrum and of the approximated root-filled bicuspid pericementum with dimming of the antrum.

FIG. 1

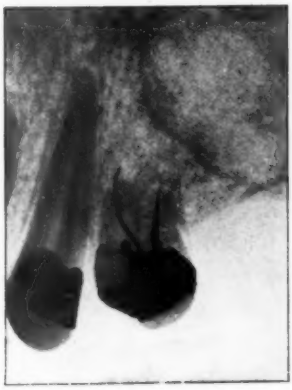

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

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FIG. 6.

FIG. 7.

FIG. 8.

FIG. 9.

FIG. 10.

FIG. 11.

FIG. 12.

FIG. 13.

FIG. 14.

FIG. 15

FIG. 16.

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Mr. F. St. J. Steadman

said he would confine his remarks to the subject of apicectomy. Mr. Colman had said this was an empirical method of removing a certain portion of necrotic root. His (the speaker's) object in performing the operation, however, was not that. He considered that was the last quarter of an inch of the root canal which mattered, and which was so difficult to treat adequately. During the last few years, in the course of which dental surgeons had had the help of radiographs, a great number of pulpless teeth had been examined and in a large number of these it had been found that the fillings had not reached their apices, and that there were infected areas around them in consequence. Hess had demonstrated the fact that there was usually not a single root canal, but that near the apex there are several small branching canals which were impossible adequately to treat. It was this lack of adequate treatment of the branch canals which was the cause of so many failures. He therefore entirely removed this portion of the root in which these branching canals were likely to exist. In performing apicectomy an incision was required which was long enough to give proper access to the apex of the infected tooth. He was convinced that when this was done the area could be effectively treated. The radiograph of the tooth he depicted on the screen was taken six months after excision of the apex of the root, which had had a large infected area round it. It was a case of a girl, aged 19, who had no other gaps in her mouth, and in whom therefore it was highly desirable to save the tooth if possible. Mr. Colman had advanced several reasons why pulpless teeth should be saved under certain circumstances, if it were possible; this was a case in point. The radiograph exhibited showed clearly that there was definite regeneration of bone round the excised portion of the root, which could not be distinguished, radiographically, from ordinary healthy bone. He (Mr. Steadman) thought that teeth like this were capable of being saved by apicectomy without detriment to the health of the patient.

Dr. Norman Grellier

said that pulpless teeth should not be retained, as radiographs often showed that they were the seat of apical infection, even without local signs or symptoms. The dentist did as much to produce as to remove infection, since in the majority of these teeth the pulps were removed during filling. Only under the most extenuating circumstances should teeth be rendered pulpless, and then they should be periodically radiographed.

Dr. A. C. Jordan

said there was nothing he could usefully add, regarding the radiological aspect of the discussion, to what had already been said. The radiologist, in his opinion, should not suggest treatment, but should be content to describe and report carefully what shown by his films.

Section of Ophthalmology.

President—Mr. Ernest Clarke, C.V.O., F.R.C.S.

[*June* 8, 1928.]

Lysozyme: An Antibacterial Body present in Great Concentration in Tears, and its Relation to Infection of the Human Eye.

By Frederick Ridley, F.R.C.S.

(*Research Student in the Institute of Pathology and Research, St. Mary's Hospital.*)

Introduction.

Lysozyme, which was described by Fleming in 1922, is an enzyme which is able to destroy not only the non-pathogenic bacteria, but also most of the organisms pathogenic for man and the lower animals. It is very widely distributed in Nature, being found in all animal tissues and tissue fluids and in most of the secretions. It is present in high concentration—in such concentration, indeed, as is destructive for many of the pathogenic bacteria in the tears, polymorphonuclear leucocytes, nasal mucus and sputum of man. This being so, it must be of the greatest importance in the defence of the normal individual against bacterial invasion. The occurrence of a marked fall in the lysozyme concentration in disease (such as I have been able to demonstrate in the tears in infection of the eye) may throw new light on the cause, natural means of cure and possible treatment of bacterial infection.

Previous Work on Lysozyme.

Historical.

Since 1902, Conradi, Meissner, Laschtschenko, Gengou, Turro, and Kuttner,[1] have described antibacterial substances which they have extracted from tissues and leucocytes. A careful examination of their work shows that in no case were they dealing with the substance lysozyme. This was first described by Fleming who, having isolated an organism which he called *Micrococcus lysodeicticus* (signifying capable of being dissolved), observed that a saline suspension of this organism was quickly dissolved by a very high dilution of normal nasal mucus.

He also found that when a drop of nasal mucus was placed on the surface of an agar plate, this organism, *Micrococcus lysodeicticus*, would not grow in that area however thickly it was planted. He gave the name lysozyme (lytic enzyme) to the substance responsible for this remarkable phenomenon.

Soon after these initial observations were made, Fleming was joined by Allison and, when no special reference is made, the research quoted in the first section of this paper was carried out jointly by them. I take this opportunity of thanking Dr. Fleming for the help and advice which he has always given to me in my own work in connexion with lysozyme.

[1] Conradi, *Beitr. f. Chem., Physiol. und Pathologie*, 1902, i. Meissner, *Zeitschr. f. Hygiene und Infektionskrankheiten*, 1912, lxxii. Laschtschenko, *Zeitschr. f. Hygiene*, 1909, lxiv. Gengou, *Annales Institut Pasteur*, 1922, xxxv. Turro, *C. R. Soc. Biol.*, 1921, clxxxiv. Kuttner, *Proc. Exp. Biol. and Med.*, 1921, xviii.

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Properties of Lysozyme.

All its properties point to lysozyme as being an enzyme.

In the pure state (as obtained by Wolff) it is a yellow amorphous powder which is easily soluble in water and saline solution. It is not destroyed by desiccation below 40° C. and retains its potency for many months, as is shown by the fact that it is present in large quantities in commercial egg white. It is destroyed by temperatures exceeding 75° C. in a short time: it acts most rapidly at 60° C., but very efficiently at body temperature. A trace of free acid or alkali inhibits its action, and any excess quickly destroys the enzyme. Lysozyme is an electro-positive or electro-neutral colloid and is adsorbed by many bodies in suspension. It cannot be filtered in the ordinary way. When the filter is saturated, however, the enzyme will pass through unhindered. It will not pass through parchment or collodion membranes, and therefore may be purified by dialysis. Peptic and tryptic digestion have no effect on the enzyme.

Wolff's work on its chemical nature shows that lysozyme gives none of the reactions for protein and, in fact, contains neither nitrogen, phosphorus, nor sulphur.

Effect of Lysozyme on Injection into the Rabbit.

Wolff has shown that lysozyme is fixed by lecithin and fats as an adsorption compound. This property accounts for the disappearance of lysozyme on intravenous or subcutaneous injection into an animal, although less than 4 per cent. is excreted in the urine. By suitable means the greater part of the lysozyme so injected can be recovered, and Wolff regards fixation by fats as the probable means of storage of the enzyme in the body.

Following upon this he showed that, although a watery extract of egg yolk contains only a trace of lysozyme, the yolk actually contains as much as egg white itself.

Wolff has also shown that no anaphylactic phenomena are to be observed in response to injection of lysozyme into an animal. On subconjunctival injection only a slight redness lasting twenty-four hours is seen.

The Phenomena of Lysis.

Lysozyme differs from other bactericidal substances, such as exist in the blood-serum, in that it is not itself used up in producing lysis of *M. lysodeicticus*. If a large amount of solid culture is added to a small amount of very dilute lysozyme solution the whole becomes clear and more organisms may be added until the mixture becomes gelatinous. If this gelatinous mass is now dissolved in normal saline the lysozyme titre is found not to be diminished.

The most striking property of lysozyme is its ability to produce lysis of an opaque suspension of the *Micrococcus lysodeicticus* and certain other bacteria. In this process a previously opaque suspension becomes perfectly clear with remarkable rapidity. As seen under the microscope the organisms swell up, lose their outline and disappear. If partially lysed organisms are stained, only minute particles not unlike the granules of the polymorphonuclear leucocyte are seen. As lysis proceeds these, too, disappear. The enzyme therefore produces complete disintegration of those organisms which it attacks by lysis. It has also been shown that it is capable, in high concentrations, of killing pathogenic bacteria without obvious lysis.

The Power of Lysozyme as an Antiseptic.

Some idea of the power of lysozyme may be conveyed by the observation that 1 grm. of the pure enzyme would confer the characteristic property of dissolving a suspension of *M. lysodeicticus* upon 100 million gallons of normal saline.

Lysozyme (tears) was compared with a large number of antiseptics in dilutions which can be employed therapeutically. To do this small discs of filter paper, each soaked in an antiseptic, were embedded in agar, and the plate planted thickly with *M. lysodeicticus*. No antiseptic tested compared with the enzyme in its power to inhibit the growth of this organism.

Estimation by Lysis.

The rapidity with which lysis proceeds depends upon the hydrogen-ion concentration, the salt content and the temperature. This offers a method of estimating the amount of lysozyme in a given solution, in that we may state the highest dilution of the material to be tested which is capable of completely clearing a standard suspension of *M. lysodeicticus* at a constant temperature in a given time.

Lysozyme Content of Various Tissues and Secretions.

Using this method of estimation, the following results for different tissues and secretions were obtained. Some lysozyme was found in all the animal tissues and secretions examined, except cerebro-spinal fluid, urine and fæces. Some vegetables, notably turnips, and the white of eggs, both fowl and fish, contain the enzyme.

In man it is present in great concentration in leucocytes and cartilage, and in tears, nasal mucus, and sputum. Taking the concentration in tears as 100, the proportion may be represented roughly as follows: tears 100, leucocytes 100 +, sputum 33, nasal mucus 33, saliva 1, and cartilage, 10. Egg white contains about 200 units. Among pathological fluids pus, alone, contains a considerable amount of lysozyme, about 5 units. It is possible, in view of Wolff's work on the fixation of lysozyme by lecithin and fats, that estimations of the enzyme extracted from tissues by water indicate only the available lysozyme and not the total lysozyme content. No investigation of the latter has been made.

The Action of Lysozyme on Bacteria.

Much work has been done on the action of lysozyme on bacteria. The effects produced may be divided into killing with lysis; killing without demonstrable lysis, and inhibition of growth—depending upon the concentration of the lysozyme and the resistance of the particular organism to its action. Fleming showed that when material containing lysozyme is placed in a trough cut in an agar plate, and this is flooded with a second layer of agar, lysozyme will diffuse through the agar, and a zone of lysozyme-containing medium will be formed. If, now, *M. lysodeicticus* is thickly planted all over the plate, the following results are to be observed on incubation. In twenty-four hours a clear zone, adjacent to the lysozyme, appears in the midst of a dense growth of the organism. In this zone it appears that all the organisms have been destroyed—the zone of primary lysis. In the course of several days a second clear zone appears which spreads up to two centimetres from the focus of lysozyme. This is called the zone of secondary lysis. It is due to a slow diffusion of lysozyme which dissolves the massive culture which grew in that area in the first twenty-four hours. After two or three weeks a remarkable change takes place. A few small isolated colonies appear in the zones of primary and secondary lysis. These, on examination, prove to be *M. lysodeicticus*, and it is found that they are very much more resistant to lysis than the original organisms—as much as thirty times. This property is maintained on subculture on ordinary media for a very long time, and it must be presumed that in an ordinary culture of the organism there is a very small proportion of organisms so resistant to the action

of lysozyme that they will survive in a state of inhibition. The fact that such resistant bacteria retain this property on subculture indicates a way in which an organism, previously destroyable by a given concentration of lysozyme, might acquire the power not only of withstanding that concentration but of growing in spite of it. It is not possible, however, to develop this power indefinitely, and after growing a strain of *M. lysodeicticus* in the highest lysozyme titre it would tolerate for a period of ninety-one weeks, its resistance was only eighty times that of the normal coccus. The ability to achieve a high lysozyme resistance relative to that of the normal organism has been demonstrated for many organisms, including *Streptococcus fæcalis*, which, although destroyed by a dilution of about one in 400 of normal tears, nevertheless can become pathogenic for man. Thus it is clear that an organism which is unable to withstand the concentration of lysozyme present in a given tissue or secretion could never invade that tissue or secretion. This gives a broad division, so far as lysozyme is concerned, into pathogenic and non-pathogenic organisms.

If we establish what is the "normal" content for a particular tissue or secretion we may further subdivide all organisms into three groups: (i) those which are incapable of developing resistance to the normal lysozyme content; (ii) those which, normally not resistant, may develop resistance to a normal lysozyme content, and (iii) those which in ordinary culture contain so many resistant individuals that they will grow despite a normal lysozyme content. Thus, we may say for a particular organism whether or not it could ever invade a tissue area of which the lysozyme titre is known.

A hundred and four strains of air-borne bacteria, mostly cocci, but containing a few bacilli and yeasts, all of which were presumably non-pathogenic, were isolated from plates which had been exposed in the laboratory. The lysozyme resistance of all these organisms was tested, and it was found that 75 per cent. were dissolved by $\frac{1}{800}$ tears or $\frac{1}{100}$ sputum. The exceptions were mainly moulds and yeasts. The coincidence of non-pathogenicity and ready lysis is strikingly demonstrated by this investigation.

The work which has been done on pathogenic organisms divides itself naturally into investigations by (1) observation of bactericidal and bacteriolytic power, and (2) observation of growth in medium containing lysozyme. The first work was done on fæcal streptococci, for which it was shown that sixteen out of nineteen strains showed definite lysis by 1 in 100 tears. Fleming investigated a large number of pathogenic organisms, using lysis by $\frac{1}{100}$ to $\frac{1}{1000}$ tears as his test. He showed that sixteen out of twenty-two strains of fæcal streptococci, and two out of four strains of staphylococci, showed some lysis, but failed to observe any effect for the other pathogenic bacteria. He made the striking observation that the *Bacillus abortus* of Bang, and the *Bacillus pseudo-tuberculosis rodentium* showed definite lysis. These organisms are culturally and serologically identical with *M. melitensis* and *B. pestis* respectively. Both the latter organisms show no lysis and are very pathogenic for man, whereas the former organisms, which show lysis, although pathogenic to some animals, are harmless to man. This investigation by lysis was continued, using $\frac{1}{20}$ egg (equivalent to $\frac{1}{10}$ tears) as the source of lysozyme, and incubating suspensions of various organisms in this medium. Slight lysis was now shown for *B. anthracis*, *B. pyocyaneus*, *B. paratyphosus* A and B, *B. aertrycke*, *B. dysenteriæ* (Flexner), *Streptococcus mutans* and *S. fæcalis*—the last-named showing very marked lysis.

Wolff employed higher concentrations of purified lysozyme, and showed that at a concentration equal to twenty times that of egg-white *B. anthracis* was completely dissolved after two hours' incubation.

Using purified lysozyme in concentrations equivalent to between 1 : 5 and 2 : 1

egg-white, he showed that the staphylococcus, hæmolytic streptococcus, *B. anthracis*, meningococcus, gonococcus and tubercle bacillus exhibit marked killing, but failed to demonstrate any action on *Streptococcus viridans*, pneumococcus, *B. diphtheriæ*, or any member of the coli-typhoid group.

He observed that while one-fifth egg concentration had no action on several strains of staphylococci, a concentration equal to egg-white had a definite bactericidal power.

The inhibitory action of lysozyme was investigated by Fleming and Allison, by incubating a dilution of a broth culture to which one-tenth egg-white had been added, and comparing the number of organisms which grew on explanting, after three to six hours, with the number which grew on explanting, from a control culture, in broth to which no egg had been added. They were able to show marked inhibition for staphylococci, *Streptococcus pyogenes*, *Bacillus coli*, *anthracis* and *typhosus*.

This work shows that most of the pathogenic bacteria are affected by lysozyme if an adequate titre is employed.

The Relation of Lysozyme to Bacterial Infection.

I was led to investigate lysozyme by the fact that cultures obtained from cases of keratitis and conjunctivitis lost virulence on repeated subculturing on ordinary media. By employing a suitable technique it was possible at first to transmit these diseases to rabbits in all the cases investigated. The fact that where a rabbit's eye remained infected with the same organism for a long period (four months in one case), the cultures obtained were always virulent, suggested that there might be some property in tears, as distinct from ordinary media, which maintained the virulence of the infecting organism. Lysozyme afforded a probable explanation of this. My first investigation was to see whether an organism which had lost virulence in the manner described had lost its resistance to lysozyme. Agar, to which had been added a known amount of tears or egg-white, was used as medium and this was planted with suitable dilutions of a broth culture of the organism (a staphylococcus) to give countable colonies. Very few colonies grew on egg-agar plates containing one-quarter egg. The organisms which did grow, however, were now virulent for the rabbit's eye. Several strains of staphylococci were tested, using plates containing serial dilutions of egg-agar, and it was found that even virulent organisms, such as hæmolytic staphylococci recently isolated from a boil, showed complete inhibition for twenty-four hours in a concentration of lysozyme equal to half tears, although in dilutions of less than one-quarter tears the cocci grew uninfluenced by the lysozyme. After forty-eight hours, 60 per cent. of the organisms planted had appeared as very attenuated colonies in dilutions up to the equivalent of normal tears, but beyond this point the number of colonies surviving was progressively smaller. After several days, 60 per cent. of the organisms grew up to a concentration equivalent to twice tears, showing partial killing and partial inhibition for virulent staphylococci. A similar result was obtained for hæmolytic streptococci. It appeared from this that a considerable fall in the lysozyme content of tears might enable staphylococci and streptococci to infect an eye which, had it had a normal concentration, would have been relatively immune. To test this, the following experiment was carried out. Normal human tears, which differ from normal saline only inasmuch as they contain lysozyme and a trace of albumin and globulin, were adapted by the addition of 10 per cent. of human serum, or 10 per cent. peptone, to form a suitable medium for the growth of various bacteria, using similarly adapted normal saline as a control. The tears so modified

were diluted with similarly modified saline in the proportions of three-quarters, one-half, and one-quarter. Fifty c.mm. of each of these dilutions, and of the control, was taken up in capillary pipettes and mixed with 2·5 c.mm. of a suitable dilution of a twenty-four-hour broth culture of the organism to give countable colonies on explanting 2·5 c.mm. of the mixture on to ordinary media. The same number of colonies was obtained on immediate explanting in both whole tears and in the control, but after six hours' incubation no colonies grew of any of the organisms affected, viz., staphylococci (three strains), streptococci—hæmolytic and fæcal (two strains), pneumococci and cholera. This means that the cultures employed, all of which, except cholera, had been recently isolated, contained less than one-half per cent. of organisms which were not actually killed by the lysozyme concentration of tears. At twelve hours a number of surviving organisms grew, even in whole tears, but in no case was the growth comparable with the control. The strains of *B. coli* and *typhosus* tested showed no effect and grew normally in six hours. The result obtained for pneumococci (which were at their maximum virulence for mice) was striking, since they grew normally in three-quarter tears, but were killed by whole tears. That the same number of pneumococci was introduced into the whole tears is proved by the fact that the same count was obtained in this pipette and in the control on immediate explanting.

Boiling the tears destroys this bactericidal power completely, and this, therefore, cannot be due to the salts contained in the tears as has been suggested (see fig. 1, p. 64).

It follows from these experiments that an eye whose tears contain a normal concentration of lysozyme is immune to infection by the staphylococci, streptococci, pneumococci, or cholera tested. It will be recalled that among other pathogenic bacteria the gonococcus, meningococcus and *B. anthracis* are readily killed by a concentration of lysozyme equal to that of tears. Thus, normal tears present a barrier to infection by any of these organisms. The tubercle bacillus which is not dissolved is nevertheless killed by a concentration equal to twice tears, as was shown by Wolff.

The Importance of Concentration of Lysozyme.

All the observations made point to the concentration of lysozyme in normal tears, sputum, nasal mucus and leucocytes, as being only a fraction greater than that which is necessary for lysozyme to act effectively upon the pathogenic bacteria. Where the body tissues are not protected by an efficient blood-supply, as in cartilage, they themselves contain a high titre. The eye and respiratory tract produce secretions which contain an adequate concentration of lysozyme. Since concentration is an essential factor in the action of lysozyme, and since for the destruction of the pathogenic bacteria the normal concentration appears to be just adequate, an accurate means of estimation had to be evolved to detect much smaller differences than had been observed hitherto.

Estimation of Lysozyme.

The technique which I now employ is as follows: A dilution of the material to be tested is made into broth. From this a series of broth dilutions diminishing by one-fifth is made in small glass tubes of about 2 c.c. capacity contained in racks of ten. To each of these tubes is now added the same volume of melted agar (2 per cent.) at a temperature up to 60° C. which will not coagulate any protein present. These broth-agar dilutions are thoroughly mixed and then "sloped." Each tube thus forms a small agar slope whose lysozyme concentration expressed as a dilution of the material tested is known.

Each of these slopes is now planted with the same amount (about 1 c.mm.) of a saline suspension of the test organism (twenty-four hours culture), adjusted to correspond exactly with a Nephelometer No. 1 suspension, and then incubated for eighteen to twenty-four hours. The test organism which I use is not *M. lysodeicticus*, but an organism which was isolated from the air. It is a Gram-positive cocco-bacillus and grows luxuriantly on all ordinary media. It occupies an intermediate position as regards its susceptibility to lysis, growing at a dilution of 1 in 3,000 tears, as tested by the above technique as contrasted with 1 in 40,000 for *M. lysodeicticus*. The organism gives a sharp end-point—one tube showing no growth and the next growth indistinguishable from that on ordinary agar. Using this method of estimation a factor of error not exceeding 10 per cent. is easily obtained and the method is capable of giving results accurate to 5 per cent.

The Unit of Lysozyme.

Normal human tears, which are constant in their lysozyme content and easily obtained, afford a suitable standard of concentration. Tears have the advantage, as compared with egg-white, that they contain only a trace of protein. The lysozyme content of all the material tested has, therefore, been expressed as a percentage of that of normal tears.

The Lysozyme Content of Leucocytes.

The remainder of this paper will be devoted to a consideration of the actual concentration of lysozyme present in normal leucocytes, and in tears in health and disease.

Fleming and Allison first showed that the blood-serum contains lysozyme as tested by lysis of *M. lysodeicticus*. They showed also that the polymorphonuclear leucocytes contain lysozyme in very high concentration. This was substantiated by the observation that bacteria, notably various strains of fæcal streptococci, which had been made resistant to lysozyme, had thereby acquired a striking resistance to intracellular digestion in leucocytes, and to the bactericidal power of whole blood as tested by the slide-cell technique.

Employing the solid medium technique of estimation which has been described but using *M. lysodeicticus* as the test organism in order to obtain a reading for very small concentrations, I was able to show that as contrasted with 48,000 units for normal tears the following values may be given for divisions of whole blood (the corpuscles of which had been disintegrated by freezing): whole blood 32 units, serum 5 units, defibrinated blood 30 units, defibrinated deleucocyted blood 4 units. It will be seen from a consideration of these figures that when the leucocytes of the blood are destroyed and their lysozyme diffused into the blood, they contribute not less than $30 - 5 = 25$ units of lysozyme to it.

Since the polymorphonuclear leucocytes represent roughly $\frac{1}{2000}$th part of the blood volume, the concentration of lysozyme within the leucocyte must be $2{,}000 \times 25 = 50{,}000$ units—that is about the same as normal tears.

The Importance of the Normal Lysozyme Content of Leucocytes and Tears.

The normal concentration of lysozyme in the tears has been shown to be very little in excess of that necessary for the destruction of the pathogenic bacteria, and this applies to the lysozyme content of the leucocyte also.

The demonstration of a fall in the lysozyme content in disease and of a subsequent rise upon recovery would be very significant. This has been observed

in estimations of the lysozyme content of tears, but no work has yet been done in respect of leucocytes. Employing the technique which I have described, the lysozyme content of the tears has been estimated for each eye in more than 100 cases.

Method of Investigation.

In each case a flow of tears was provoked by applying a drop of lemon juice with a sterile platinum loop to the conjunctiva of the lower lid at the inner canthus. The tears were collected in a Wright's blood capsule from the outer canthus. A trace of lemon does not influence the reading obtained. The capsules were now labelled and sealed and kept in the ice-chest, pending estimation. In this way the lysozyme, even in tears from an infected eye, may be preserved unchanged for weeks.

Causes of Reduced Lysozyme in Tears—Epiphora.

A fall in the lysozyme concentration of the tears always accompanies epiphora lasting more than a few hours. All conditions which lead to such a period of epiphora thus expose the eye to immediate danger of infection. In five cases of foreign body retained in the eye for more than three days the concentration in the affected eye was less than 45 per cent., the other eye being normal. Two cases are interesting as showing the significance of such a reduction. In each the foreign body was a particle of stone which had been retained for three days and had produced acute epiphora. In one case the eye was injected only and cleared up at once when the foreign body was removed; in the other acute conjunctivitis and hypopyon ulcer had developed and this was associated with obstruction of the duct on that side and an old-standing chronic dacryocystitis. The organism infecting the sac had not previously invaded the conjunctiva, but did so at once when the lysozyme titre fell. The patient recovered under treatment, and the lysozyme of the affected eye returned to normal, although the sac infection persisted.

This case indicates, firstly, that a chronic infection may invade a previously immune tissue area if the lysozyme titre in that area falls below 50 per cent., secondly, that the lachrymal gland is able to maintain the titre of its secretion to only a limited extent in epiphora, and thirdly, that recovery from an infection is accompanied by a return to the normal titre of lysozyme.

In sixteen cases of conjunctivitis, both acute and chronic, including several cases of angular conjunctivitis, the infected eye was found to have a reduction of lysozyme to between 30 and 60 per cent.

The unaffected eye was found in most cases to have a normal titre. This reduction of the lysozyme content in the tears of infected eyes was observed in all the cases investigated, including cases of trachoma, corneal ulcer, hypopyon ulcer, phlyctenular conjunctivitis and keratis, and interstitial keratis.

Nine cases of monocular infection having a normal concentration in the unaffected eye, were followed up by repeated estimation, and in each case recovery was associated with a return to normal titre in the affected eye.

In three cases of obstructed lachrymal duct without conjunctivitis the concentration was normal in the affected eye.

General Causes of Lysozyme Reduction.

Finlay observed that in growing rats deprived of Vitamin A a condition of keratomalacia developed, and that this was associated with a fall of lysozyme in the tears. He also observed that if a drop of human tears was introduced into the conjunctival sac from time to time, the onset of this condition could be delayed.

In six cases of early phlyctenular disease, and in four cases of recent interstitial keratitis, it was noticed that the lysozyme titre of both eyes was below 55 per cent. of normal, although in two of the former and two of the latter only one eye was affected. This low lysozyme titre in the unaffected eye appears to be confined to these two conditions, and may be due to the general ill-health which is associated clinically with them. This would account for the regularity with which these diseases attack both eyes as had happened in six cases of phlyctenular disease and nine cases of interstitial keratitis examined.

The low lysozyme titre observed in these cases may account for the clinical fact that these eye diseases do not recover until the underlying systemic disease or deficiency has been effectively treated.

This is of importance in forming a conception of the action of lysozyme in the natural cure of these eye diseases, since it is obvious that local treatment could only raise the titre of the affected eye to that of the unaffected eye, and in cases having a low titre in the unaffected eye this would be inadequate to bring about recovery from the infection.

Further, in five cases of interstitial keratitis which had received thorough general treatment and were well at the time of examination, the titre in both eyes was over 80 per cent., and in one case, examined three months after complete recovery and when the full course of injections had been given, was 100 per cent. in each eye.

I have observed recently two cases of typical interstitial keratitis—the first (L. S.), in a woman of 33, having a positive Wassermann reaction and typical signs of congenital syphilis. The right eye was first affected and it was noticed, as in other cases, that the lysozyme content of the apparently normal eye was almost equally reduced, 55 per cent. and 60 per cent. respectively. Within a month the second eye became involved, and both eyes are running the usual clinical course. The epiphora common to all acute eye infections has been well controlled by atropine, but the lysozyme content after two months has never risen above 70 per cent. in either eye.

The other case (C. K.) involved the right eye in a boy of 15. He showed no signs of congenital syphilis, and his Wassermann reaction has been repeatedly negative. He was exceptional in that the lysozyme titre of his left eye was from the first normal (see chart). The right eye watered very freely, and its titre remained at about 45 per cent. for six weeks. At the end of this time the epiphora which followed the instillation of atropine was so marked that a change was made to scopolamine. From this time the epiphora became less, the eye improved rapidly both as regards the conjunctivitis and the corneal condition, and within four days the titre of the affected eye rose to normal (100). This has been maintained for three weeks, and the eye is now white and the cornea clearing rapidly. This case, as contrasted with the last (L. S.), shows the significance of the titre of the normal eye in prognosis. It also shows in a striking manner the importance of lysozyme in recovery from infection and its relation to epiphora. It may be that the estimation of the lysozyme titre of the normal eye in " interstitial keratitis " in its early stages is a useful guide to treatment and prognosis.

The Clinical Importance of the Titre in Cases of Keratitis.

To treatment and prognosis might possibly be added " diagnosis," for the reduction of lysozyme in the normal eye was seen in several cases having positive signs of congenital syphilis, and was not observed in this case which showed no signs of congenital syphilis. It may be considered heretical to attribute interstitial keratitis to a local infection other than the spirochæte, but there is evidence that this may be so.

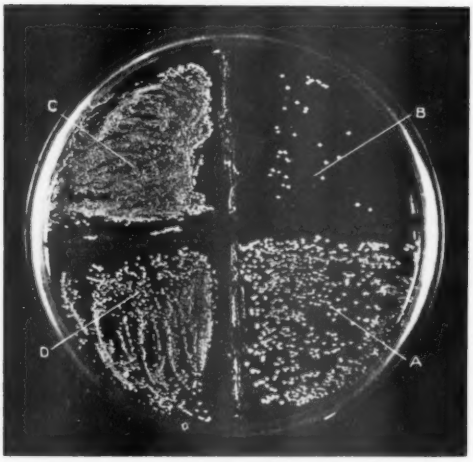

FIG. 1.—SHOWING THE BACTERICIDAL POWER OF TEARS AND ITS DESTRUCTION BY BOILING.

(*A*) Control count of staphylococci implanted in *B*, *C*, and *D*.
(*B*) Number of cocci surviving after six hours' incubation in tears and 10 per cent. serum—explanted on to agar.
(*C*) Cf. *B*, but using tears which had been boiled, the lysozyme being thereby destroyed.
(*D*) Cf. *B* and *C*, but using normal saline instead of tears.

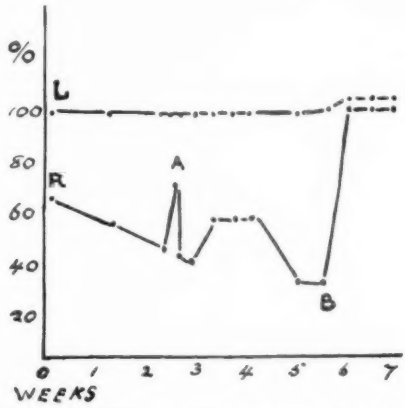

FIG. 2.—CHART SHOWING THE VARIATIONS OF LYSOZYME TITRE IN A CASE (C K.) OF INTERSTITIAL KERATITIS.

Left eye, normal, thirteen estimations are shown, in each of which the titre is 100 per cent. (normal tears being estimated as a control in each case).
Right eye, affected (see text).
(*A*) Rise following direct injection of the lachrymal gland with atropine.
(*B*) Point at which treatment was changed to scopolamine—an immediate and permanent rise in titre followed. Epiphora was very marked up to the point *B*, except for a few hours at *A*, and ceased after the point *B*.

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The Action of Atropine in the Eye.

It has been observed consistently that epiphora is associated with a fall in the lysozyme titre. When epiphora ceases the lysozyme titre rises, and this has been associated in every case with clinical improvement in the eye condition. It is well recognized that epiphora is much reduced by the instillation of atropine in eye affections, especially of the cornea, and this is commonly attributed to its action as a mydriatic and to its "putting the eye at rest." It is more than this, however, for even painful stimulation of the conjunctiva in an eye thoroughly under the influence of atropine, evokes only a small flow of tears as compared with the normal eye. The lachrymal gland itself must be partially paralysed by atropine instilled into the conjunctival sac. In one case (C. K.), showing acute epiphora and a titre of only 45 per cent., the gland was injected directly with atropine sulphate—epiphora ceased at once, and the titre rose to 70 per cent. in a few hours (see chart). It may be that the action of atropine in producing clinical improvement in infections of the eye, is due to its action in reducing epiphora, and in consequence producing a rise in the lysozyme titre: this reduction of epiphora being due partly to its direct paralysing action on the lachrymal gland, and partly to its action as a mydriatic, whereby reflex stimulation of tear secretion is diminished.

Possible Use of Lysozyme in Treatment.

Since lysozyme is tolerated by the tissues in concentrations as great as forty times tears (as has been shown by Wolff), the possibilities of its application in the treatment of bacterial infections cannot be overlooked. Work is proceeding on this question both here and abroad. The preparation of the enzyme in a pure state presents many great difficulties and demands a laboratory specially equipped for the purpose.

As is quoted by Fleming in his first paper on lysozyme, Metchnikoff in his treatise on "Immunity and Infectious Disease," says "Nature, to protect the skin and mucous membranes, does not use antiseptics. The fluids which bathe the surface of the mouth and other mucous membranes are not bactericidal, or very imperfectly so."

It is clear from the work which has been done upon lysozyme that this view, which is still generally held, must be changed.

Nature *does* provide, especially in the tears, a very efficient antibacterial substance, lysozyme, to which must be attributed an important rôle in the prevention of, and recovery from, bacterial infection.

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Discussion.—Dr. ALEXANDER FLEMING said that he could say little, as Mr. Ridley had summarized all the work that he (the speaker) had done on the subject. This anti-bacterial substance, which was inhibitory or which dissolved bacteria, according to the degree of concentration in which it was used, was found in places in the body where it was wanted in the nose, the sputum, and in those parts which were constantly being exposed to microbes from the air. The general conception about the secretions and their beneficial action was

that they washed the microbes away, and this they did to a certain extent. But he wondered whether his hearers had tried to wash bacteria out of a test-tube; it required a good deal of water to do so. It was doubtful whether the degree of washing in the case of the eye was worthy of consideration. But there was no doubt that tears had the power of dealing with the ordinary microbes met with, and, in full concentration, were able to deal with the ordinary pathogenic microbes in the eye in a floating state.

It might be said that the lysozyme had also a general antiseptic action. It had been shown that leucocytes had this substance in concentration sufficient to deal with the ordinary pyogenic microbe. The process of phagocytosis was not yet clearly understood. It had been attributed to a tryptic ferment, but ordinary trypsin had no effect on the ordinary pathogenic microbe. There was considerable evidence to support the idea that lysozyme was in some way connected with intracellular digestion.

Mr. Ridley's work on the clinical condition carried the work a stage further, and his observations should have far-reaching results. Egg-white was a good means of getting lysozyme quickly. Wolff, in Holland, isolated this ferment from egg-white, and obtained it many times stronger than the naturally occurring secretion. Therefore it was possible that in the future this ferment would be at the disposal of the profession in a highly concentrated form, in which it could be used for the treatment of infections.

Mr. ERNEST CLARKE (President) said that a vast amount of new information had been presented that atropine was given hypodermically to check secretions in most of the glands, and one could understand that it did inhibit the lachrymal gland. It was also of interest to learn that epiphora, if allowed to go on, was damaging by lowering the titre.

Section of Neurology.

President—Edwin Bramwell, M.D.

[*April* 19, 1928.]

Clinical Meeting held at the West End Hospital for Diseases of the Nervous System.

Cerebellar Ataxia.

By C. T. Potter, M.D.

B. W., aged 16. Complains of unsteady and staggering gait—occasional headache and difficulty in speech.

History.—Quite well until sixteen months ago. It was then noticed that his right leg was thrown out to the side in walking. He soon became unsteady on his legs and walked with staggering gait; speech also became difficult—slow and indistinct. About the same time (sixteen months ago) he had severe and frequent frontal headaches lasting about ten minutes at a time; these have recurred on an average of once a month and are not accompanied by vomiting.

Previous History.—Accident—fall on forehead—at 5 years; unconscious ten minutes.

Family History.—No history of any similar case in family or relatives.

Present Condition.—Pupils: left larger than right; both react normally. Fundi normal. Marked nystagmus, especially lateral. Slight weakness left internal rectus. Other cranial nerves normal. Sensation normal. Slight intention tremor, right greater than left. Muscular tone good. Gait ataxic on a wide base; flat feet. Arm-jerks present and equal. Knee- and ankle-jerks present and equal. Plantars flexor. Abdominals normal. Incoördination on finger-nose test, also on heel-knee test, right greater than left. Romberg negative.

Cerebro-spinal fluid: cells 3 per c.mm.; total protein 0·03 per cent. Wassermann and Lange reactions negative. Blood: Wassermann reaction negative.

Disseminated Sclerosis with Myasthenic Facies.

By C. T. Potter, M.D.

D. H., aged 22, complains of unsteadiness in walking, difficulty in speaking and occasional double vision.

History.—About fourteen months ago she began to drag the right foot; this was followed shortly afterwards by weakness of right leg. At the same time speech became difficult and slow. She had also had occasional diplopia on looking to extreme right or left, and during the past four months has developed tremors of the hands. No previous illnesses of importance.

Family History.—Mother had "paralysis" (the patient claims that this "paralysis" was practically the same as her own).

Present Condition.—Intelligent and with good memory. Facies: Marked nasal folds—myasthenic snarl. Pupils equal and react normally. Fundi normal. Coarse nystagmus to right and left. Right and left external recti weak. Lower jaw deviates to right. Other cranial nerves normal. Sensation normal.

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Slight intention tremor of both hands; arm-jerks present and equal. Right leg spastic and weak. Knee- and ankle-jerks increased, right greater than left. Double patellar and ankle clonus; plantars extensor. Abdominal reflexes absent. Gait spastic on a wide base. Incoördination of both arms and legs. With faradism, no myasthenic reaction was found in masseter muscle nor in muscles of arms.

Cerebro-spinal fluid: 15 small lymphocytes per c.mm.; protein 0·03 per cent. Wassermann reaction negative. Lange: 5555431000. Blood: Wassermann reaction negative.

Charcot's Arthropathy of Both Wrists.

By C. Worster-Drought, M.D.

W. H., aged 50, painter, has complained of swelling of the right wrist since 1918. The onset was gradual and without pain. About a year later the left wrist became slightly affected and the metacarpo-phalangeal joints of the left index and middle fingers began to enlarge.

Fig. 1.—Right wrist-joint.

No history of syphilis. Married twenty-seven years, with one child, now aged 25.

Physical Examination.—The right wrist is irregularly enlarged and much deformed. On the dorsum, towards the ulnar side, there is a large swelling containing some fluid with bony thickening beneath. A similar, though smaller, swelling is situated on the radial side of the joint. The enlargement is neither painful nor tender. Flexion and lateral movement at the joint are moderate and extension very limited.

FIG. 2.—Skiagram of right wrist (lateral view).

X-ray examination shows total disorganization of this joint, the carpus having almost entirely disappeared, and the lower ends of the radius and ulna being dislocated dorsally on to the bases of the metacarpal bones.

The left wrist is considerably swollen, especially towards the ulnar side, and deformed. No tenderness; flexion and extension fairly good. Skiagram shows an early stage of Charcot arthropathy, with little bone damage.

The metacarpo-phalangeal joints of the left index and middle finger are also swollen and enlarged, but freely movable; X-ray examination shows signs "suggesting early Charcot joints." In the right axilla are several much enlarged glands,

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which are neither painful nor tender. Epitrochlear glands are palpable in each arm, the left one being larger than the right.

Central nervous system: Right pupil larger than left; neither pupil reacts to light, but reacts to accommodation-convergence. Other cranial nerves normal. Some ulnar and tendo-Achillis analgesia. Arm-jerks and abdominal reflexes normal. Knee-jerks present, right slightly brisker than left; ankle-jerks normal; plantars flexor. No definite incoördination.

Blood: Wassermann reaction positive. Cerebro-spinal fluid: Sixteen lymphocytes per c.mm.; globulin increased; Wassermann reaction positive. Colloidal gold reaction shows curve of luetic type.

During the past three years this patient has been treated with courses of novarsenobillon intravenously and bismuth intramuscularly. The neurological signs and the right wrist and metacarpo-phalangeal joints remain unaltered, but the left wrist has slowly deteriorated.

Commentary.

The wrists and metacarpo-phalangeal joints are very rarely affected by Charcot arthropathy in tabes dorsalis. Thus, of 268 tabetic arthropathies collected by Chipault[1] the wrist was affected in only three cases and the finger-joints in two. Next to the temporo-maxillary joint (affected in one case of Chipault's series) the wrist- and finger-joints are those most rarely involved. Of the 268 cases, no less than 207 were arthropathies of the lower limbs (120 with knee affected, fifty-seven with hip-joint). Of other authors, Gowers[2] gives the relative frequency of the joints affected, as follows: knee forty-five, hip twenty, shoulder eleven, tarsus eight, elbow five, ankle four, and does not mention the wrist at all. Collier and Adie[3] give knee, hip, shoulder, elbow, ankle, small bones of hands and feet, spine; while R. Jones and R. W. Lovett[4] place the wrist last.

In this case, as occasionally occurs, the arthropathy of the wrist was the first indication of tabes. The only neurological signs present are the Argyll-Robertson pupils, the slight inequality of the knee-jerks and the ulnar and tendo-Achillis analgesia.

The influence of this patient's occupation—that of a painter—is almost certainly of considerable importance in determining the arthropathies of the wrists. The alternate flexion and extension of the wrist, continued over long periods, constituted the double factor of fatigue and the risk of strain. When the right wrist became incapacitated, this patient began to paint with his left hand, with the result that this joint also has become increasingly affected during the past three years.

Other features of interest in the case are the enlarged right axillary glands and the bilateral epitrochlear glands.

Note.—This case, at an earlier stage, was shown before the Clinical Section, November 12, 1926.[5]

1 Chipault, A., "Traité de Chirurgie" (le Denton et Delbet), Paris, 1896, iii, 458.
2 Gowers, W., "Diseases of the Nervous System," London, 1899, 1.
3 Collier, J. S., and Adie, W. J., "Text-Book of Medicine" (F. W. Price), London, 1926, 1460.
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5 *Proc. Roy. Soc. Med.*, 1927, xx (Clinical Sect.), 192.

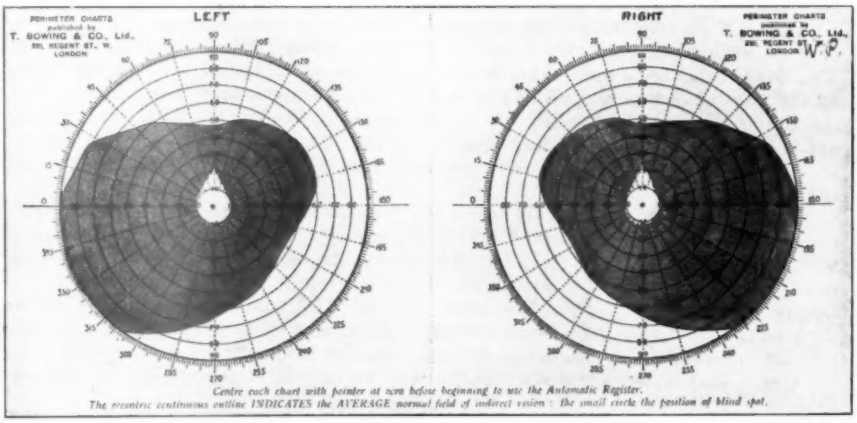
Section of Neurology 51

Bilateral Calcarine Thrombosis with Retention of Macular Vision.

By C. WORSTER-DROUGHT, M.D.

W. P., AGED 48, complains of (1) defective vision—"can see only straight ahead," and (2) occasional dizziness.

History of Condition.—One evening in February, 1919, he found, on coming out of a shop, that he walked into people on his left side (? left hemianopia of sudden onset). There had been no premonitory symptoms and until this occurrence he had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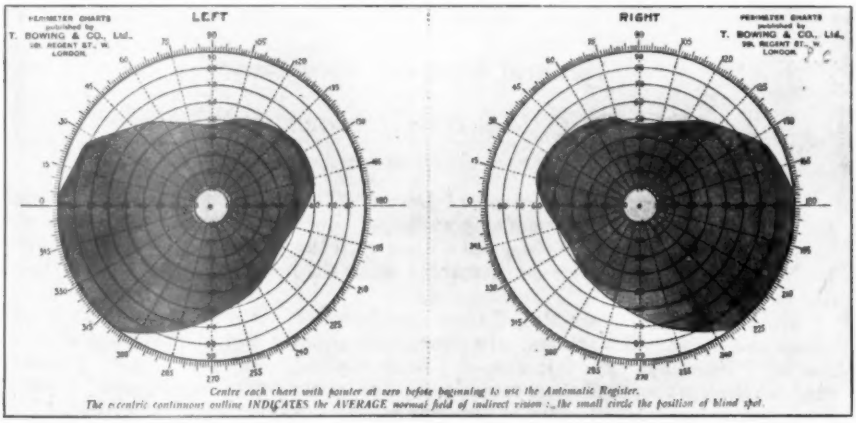
Bilateral Calcarine Thrombosis.—Visual fields.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The eccentric continuous outline INDICATES the AVERAGE normal field of indirect vision: the small circle the position of blind spot.

Bilateral Occipital Thrombosis.—Visual fields.

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felt perfectly well. On the following day he complained of slight right-sided occipital headache and occasional dizziness. There was no further change for nine months. Then, one evening during November, 1919, while walking in the street, he suddenly realized that he could see scarcely anything at all. He felt somewhat dazed and dizzy, but noticed no other symptoms. His condition has remained substantially the same since this time.

Previous History.—No previous illnesses. Prior to enlistment in 1915 he had worked at a large drapery establishment for seventeen years without a break. He served in France from May, 1916, onwards, and towards the end of that year was blown out of a gun-pit by a shell explosion, but sustained no injury or loss of consciousness. He continued his service in France until demobilized in January, 1919. No history of syphilis or alcoholism.

Physical Examination.—All peripheral (panoramic) vision absent, central vision only remaining (see charts). Eyes emmetropic; direct central vision $\frac{6}{9}$ in each eye. The visual fields have been examined on many occasions during the past eight years by different observers and always have been found substantially the same. Of the remaining (central) portion of the visual fields, in the right eye the field for red appears largest, white next and that for green smallest. In the left eye, the field for red is largest in the upper part, that for white in the lower part, and green again the smallest.

Pupils equal, right slightly eccentric; both react normally to direct light, consensually and to accommodation-convergence. Optic discs a little pale, but within physiological limits; the left disc shows opaque nerve fibres with consequent indistinct edge on nasal side; fundi otherwise normal. Occasional tendency to nystagmoid jerks on lateral deviation of eyes. All other cranial nerves normal. Arm-jerks and abdominal reflexes normal. Knee- and ankle-jerks normal and equal, plantar reflexes flexor. Sensation normal. No incoördination.

Heart and arteries normal; pulse-rate 76 regular. Blood-pressure 145 mm. systolic, 75 diastolic.

Blood yields a negative Wassermann reaction. Cerebro-spinal fluid; normal as regards cell and protein content, with Wassermann and Lange reactions negative.

X-ray examination of occipital region and sella turcica reveals no abnormality.

Bilateral Occipital Thrombosis.

By C. Worster-Drought, M.D.

Mrs. S. C., aged 72.

History.—During the afternoon of December 27 last she complained of slight occipital pain, and a short time afterwards asked her daughter to light the gas as it had "become so dark." It was then discovered that she was almost totally blind. Earlier in the day she had seen everything quite distinctly, and the evening before had been playing cards.

For the past two years Mrs. C. has complained at times of temporarily blurred vision and occasional dizziness. In June, 1926, she was seen at hospital, and a note states: "Right eye, $\frac{6}{12}$; left eye, $\frac{6}{12}$; fields normal, fundi normal, no cupping." Her daughter states that more recently she has become very forgetful and "dreamy." There are no previous illnesses of importance.

Physical Examination.—Vision in both eyes is restricted to an extremely small amount of central vision for light and bright objects only (vide charts), e.g., she can

detect a centrally placed light, a brass candlestick, or a white towel. All peripheral perception is absent. Pupils equal and regular; direct light, consensual and accommodation reflexes present in both. Early and small lenticular opacities corresponding to about 7 and 8 o'clock in both eyes. The fundi show no abnormality beyond slight tortuosity of the retinal arteries. Other cranial nerves normal. Reflexes, sensation, &c., show no abnormality. Peripheral arteries are not unduly thickened nor tortuous; blood-pressure 240 mm. systolic; 120 diastolic. Heart shows border-line enlargement, accentuated aortic sound and occasional premature beats. Urine contains no albumin, sugar nor casts.

COMMENTARY ON THE TWO FOREGOING CASES.

To account for these rare cases of loss of panoramic vision with persistence of central vision, the explanation advanced by Holmes and Lister is usually accepted, viz., that the occipital pole where macular vision is represented, derives its blood supply from two sources: (1) calcarine branch of the posterior cerebral artery which supplies the whole of the visual cortex, and (2) a branch from the middle cerebral artery. When occlusion of both posterior cerebral arteries occurs, macular vision persists owing to the branch from the middle cerebral artery being unimpaired.

In both the cases shown the vascular nature of the lesion is at once suggested by the sudden onset of the visual impairment. In the first case, the right side was affected nine months before the left, while in the second both sides were involved simultaneously. In this second case the thrombosis is clearly due to arterio-sclerosis, as indicated by the condition of the retinal and peripheral arteries, the high blood-pressure, etc.

In the first case, the ætiology is by no means clear. There is no evidence of arterial disease, syphilis or other disturbance. A benign tumour (e.g. fibroma) of the posterior end of the falx cerebri and compressing the calcarine arteries is possible, although there is no evidence in favour of such a condition. Some years ago, at another hospital, a diagnosis of hysteria was suggested in this case, but it is understood that the patient failed entirely to respond to various forms of psychotherapy. For this reason the fact is mentioned in his history that, although he was subjected to the most potent factors productive of so-called "shell-shock," he subsequently suffered from no such condition, and continued to serve in France until the end of the war. Further, some years' acquaintance with him has convinced me that he is anything but a hysterical type. In his case, the retention of fairly good central vision ($\frac{6}{9}$) is in marked contrast to the condition of the second patient, who can only distinguish lights and very bright objects when centrally placed. Presumably this could be explained on the basis that, whereas in the first case the remaining blood supply to the occipital pole is carried out through a healthy branch of the middle cerebral artery, in the second case this branch is probably atheromatous.

Case of Dyspituitarism.

By C. WORSTER-DROUGHT, M.D., and B. W. CROWHURST ARCHER, M.B.

H. S., MALE, aged 15, was brought to hospital on February 1, 1928, for investigation on account of the following symptoms: unusual size for his age, excessive obesity, difficulty in walking, attacks of vomiting, and mental backwardness.

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Previous History.—He was normal at birth; sat up at the usual age; cut his teeth at the usual time; but did not walk until nearly three years of age. Between 3 and 4 he began to grow in stature rapidly and to become much more obese than the average child of his age. At that time it was noticed that he was backward mentally. He went to school at the age of 5, but made no progress; has never learned to read or write, but has shown some interest in mechanical things. He

Comparison with normal boy of the same age.

has always been good-natured and his behaviour is invariably good. He plays with boys of his own age and indulges in the same games as they, but without any success. During the past year he has not been able to get about very well on account of "difficulty with his legs." His appetite is described as "tremendous" and he is unusually fond of sweets. Thirst is within normal limits. He frequently

suffers from attacks of vomiting at night; sleeps fairly well and shows no tendency to fall asleep during the day; seldom suffers from headaches, and sight and hearing are good. According to his father, he developed late sexually. He has had attacks of influenza, but in none of these has he ever complained of any disturbance of vision.

Family History.—The patient is the youngest and the only one living of a family of five. The fourth child died at birth following a breech presentation, and two others died of scarlet fever. All the children were big, but in the others no changes were noticed similar to those in the patient. His mother is "nervous" and has had no miscarriages or premature infants. His father is healthy; height 6 ft. and appearance is suggestive of acromegaly. He says that his father was a much bigger man than he is.

Physical Examination.—His height is 6 ft. 1 in. and weight, stripped, 19 st. 8 lb. He exhibits pronounced general obesity with fairly uniform distribution of the fat; there is slight differentiation of the shoulder and pelvis girdles, but no padding in the supraclavicular regions or on the dorsum of the hands. Hair is fine and normal in amount; in the pubic region it is scanty and conforms to the male distribution. Skin of normal texture, covered with a slight growth of fine hair, and exhibits an acneiform eruption on the face. Penis is small and somewhat retracted; testicles are descended and apparently normal. Muscular power relatively weak. Circumference measurements of limbs and body are as follows: upper arm, 14¼ in.; forearm, 13 in.; thigh, 27¾ in.; calf, 17½ in.; chest, 48 in.; abdomen, 47 in. The forehead is full and prominent; jaws large; palate arched, and the upper teeth project beyond the lower. Temperature is invariably subnormal, rarely reaching above 98° F.; heart, lungs, and abdominal viscera show nothing abnormal; pulse-rate averages 90; blood-pressure 125 systolic and 75 diastolic. Urine shows no abnormality.

Central Nervous System.—Pupils are moderately dilated, and equal, and react normally; visual fields and optic discs also normal. The functions of all the cranial nerves are normal. No disturbance of sensation or coördination. Motor power in upper limbs fair, but below normal. The two hands are constantly held in a position of flexion at the wrist, full extension at the metacarpo-phalangeal joints, hyperextension at the interphalangeal joints and approximation of the tips of the fingers to one another. Lower limbs normal, except that in standing the knees are held close together and the feet separated. Abdominal reflexes are brisk and equal, arm reflexes, knee- and ankle-jerks normal, and plantar reflexes flexor. Gait is slow and awkward with knees close together and feet separated.

X-ray examination shows the sella turcica to be rather small but scarcely outside the limits of normal; frontal sinuses unusually large, and lower jaws big and prominent. The epiphyses of the wrists are normal, but those of the fingers are united.

Cerebro-spinal Fluid.—One small lymphocyte per c.mm., normal protein content, negative Wassermann and colloidal gold reactions.

Blood.—5,490,000 red cells per c.mm. with slight anisocytosis at or about normal size. Hæmoglobin, 92 per cent.; colour index, 0·84. White cells, 8,600 per c.mm., and show a lymphocytosis involving chiefly the large lymphocytes, viz.: 67 polymorphonuclears, 15·5 small lymphocytes, 14·5 large lymphocytes, 1 large mononuclear and 2 eosinophils per cent.

The resting blood-sugar is 0·096 per cent.; the blood-sugar curve—following the oral administration of 50 grm. of glucose—is normal, rising to an apex of 0·168 per cent. in one hour, and falling to 0·106 per cent. in two hours.

Mentally, the boy is very backward; he can neither read nor write. His mental age according to the Stanford modification of the Binet-Simon scale is between six

and eight years, though for questions involving simple matters of conduct, it might be placed at the higher of the two levels. His intelligence quotient on the tests made, is about fifty.

Commentary.

This case suggests a condition of mixed dyspituitarism, viz., an increase in anterior lobe function and a diminution in posterior lobe function. Probably, the anterior lobe being enlarged and the pituitary fossa remaining fairly normal in size, the pars intermedia and pars nervosa have become compressed, with resulting reduction in functional activity. The features suggesting anterior lobe increase are: (1) rapid growth to a height of 6 ft. 1 in., and, (2) skeletal over-growth with large jaws and prominent frontal sinuses. Posterior lobe diminution is indicated by: (1) obesity, (2) fine and scanty body hair, (3) small genitalia, and (4) lymphocytosis. The tendency for the relatively inactive posterior lobe to produce increased sugar tolerance is probably counteracted by the increased anterior lobe function, resulting in a normal blood-sugar curve. There is no evidence of definite tumour formation.[1]

Motor Abnormality in a Child. Case for Diagnosis.

By I. M. Allen, M.D.

S. C., FEMALE, aged 5, was brought to hospital because she had had difficulty in walking since the age of 2 years. At this age it was noticed that she sometimes tripped and almost fell when walking. Later, movements of the legs, arms and body were associated with the difficulty in walking. The condition has become definitely and steadily worse since the onset. The child has always been strong and healthy. She was full-term; began to walk at 1 year; and began to talk at 18 months.

Family History.—The patient is the only child. Her father suffers from spasmodic torticollis, which is sometimes associated with spasmodic movements of the right arm. This condition appeared one month after he was demobilized in June, 1919, but was not severe until 1923, when it increased in severity after a pension awarded on account of malaria was discontinued. There are no other features of importance in the family history.

Physical Examination.—Gait: For the first few steps the patient walks normally, but with an evident appearance of anxiety. A sudden movement of flexion of the right knee and adduction of the right thigh then occurs invariably and appears to upset her balance. The left leg is then lifted, swung out, internally rotated at the hip and the foot brought down on the floor in that position. It appears as if she will fall, but as a rule she does not do so; sometimes she does fall. At the same time both arms are held in front as if warding off an attack—abducted at the shoulders, flexed and pronated at the elbows, slightly extended at the wrists, extended at the metacarpal-phalangeal joints and flexed or extended at the interphalangeal joints. As she proceeds, slight, slow, twisting and turning movements occur at each of these joints. At the same time, an irregular wave-like writhing of the body occurs, but is not in any way pronounced. On other occasions the arms are held behind the body; violent movements of dorsiflexion of the spine occur, sometimes slowly and sometimes rapidly, followed by a slow return to the normal position, and sometimes a single spasmodic movement of the head to one side. As

1 *Postscript.*—During the meeting it was found that this case had been under Dr. Grainger Stewart at an earlier age, and was shown before the Section for the Study of Disease in Children by Dr. Macdonald Critchley in 1925 as a "Case of Obesity, ? Pituitary in Origin" (*Proc. Roy. Soc. Med.*, 1926, xix, Section for Study of Disease in Children, 41).

a rule these movements are slow, twisting and wave-like, and in some degree rhythmical; but at times they are rapid and spasmodic.

There is spasmodic alteration of muscle tone, now hypertonus, now hypotonus predominating. At rest and in walking, sudden shock-like contractions are observed and palpated in muscles of the upper and lower limbs and of the back. The contraction comes on suddenly, may forcibly move the joint, is not repeated at once in the same muscle or group of muscles, but may occur elsewhere. It is observed especially in the muscles of the right side of the body—in the scapular muscles, the flexors of the forearm, the pronators of the forearm, the flexors of the wrist and the extensors of the fingers. In the leg it occurs in the hamstrings and adductors of the thigh. It occurs in the sterno-mastoid and results in sudden rotation of the head to one side and then to the other. In the spinal muscles it occurs bilaterally and results in dorsiflexion of the spine. Similar contractions are observed in the left forearm, in the superficial muscles of the flexor group and in some muscles of the extensor group. Occasionally they occur in the hamstrings of the left leg. These sudden shock-like contractions occur when she attempts to walk—especially in the hamstrings and adductors of the right thigh, the flexors and extensors of the wrists and the spinal muscles—and appear to initiate the rhythmical and wave-like movements which appear as she walks. Movements also occur at rest, but not so extensively, when the child is excited.

Central Nervous System.—The muscles of the neck, trunk and both upper and lower limbs are characterized by extreme hypotonia; and it is possible to bend the limbs and body at will. There are no contractures. At times the eyes stare and blinking seldom occurs; at others, blinking occurs relatively frequently. Pupils equal and dilated; react briskly to light and consensual stimulation but sluggishly to accommodation. Vision: visual fields, fundi and the ocular muscles normal. Speech and hearing are normal. Voluntary movements of the face are normal, but no emotional movements are seen. Other cranial nerves normal. All sensation normal. Upper limbs—no wasting and motor power normal and equal on the two sides. The deep reflexes vary from time to time; sometimes they can be elicited; sometimes they are present but weak. Repeated fine movements are performed equally well in the two arms, but they tire and become clumsy more easily on the right side. Lower limbs—no wasting and motor power normal. Knee-jerks present; on the right side the leg rises and falls more rapidly on percussion than on the left. Ankle-jerks present and equal; no ankle clonus. At times the knee-jerks and ankle-jerks cannot be elicited. Plantar reflex definite and constant plantar-flexion on the right side; but on the left side sometimes plantar-flexion and sometimes dorsi-flexion appears first. Abdominal reflexes brisk and equal. No spontaneous movements at rest but as the examination proceeds the shock-like contractions and twisting movements described above appear and increase in intensity. The patient stands unsteadily on a wide base and there is no increase in swaying on closing the eyes. Sphincters—normal. Cerebro-spinal fluid on withdrawal contained blood, which was more apparent after the withdrawal of the first c.c. Wassermann reaction—negative. Pulse 88; blood-pressure 90/55; heart and other systems normal.

Nine months' removal from home failed to produce any improvement and prolonged treatment by persuasion and suggestion in hospital has not met with success. On treatment with borax and bromides, the contractions are less frequent and less severe, but the movements which appear on attempting to walk are unchanged.

The probable diagnosis is one of "myoclonus epilepsy" with superimposed hysteria.

Bilateral Sixth Nerve Paresis in a Child.

By I. M. ALLEN, M.D.

D. H., FEMALE, aged 6 years, was admitted to hospital on February 20, 1928, with a history of squint in the right eye for five days, and of slight fever (99° F.) at one observation.

Previous history and development normal. Family history: There is a history of tuberculosis among the father's relatives.

Central Nervous System, February 20, 1928.—Pupils moderately dilated, equal and react normally. Vision: visual fields and optic discs normal. Slight weakness of both external recti. Nystagmoid movements of the eyeballs are present on lateral and upward deviation. There is some lack of emotional expression in the face. Other cranial nerves normal. Sensation normal. Upper limbs: tendon reflexes exaggerated and equal. Lower limbs: tendon-jerks exaggerated and equal; plantar reflexes flexor. Abdominal reflexes present and equal; gait normal; spontaneous movements *nil*; coördination normal. Temperature rose to 99° F. on one occasion after admission; afterwards normal. Cerebro-spinal fluid: nothing abnormal; Wassermann reaction negative. X-ray of skull: negative. Other systems all normal.

Subsequent Progress, February 27, 1928.—Weakness of both external recti increasing.

March 7, 1928.—Few emotional movements of face observed since admission. All arm reflexes difficult to elicit. Knee-jerks equal—rise and fall rapidly.

April 10, 1928.—Weakness of both external recti much increased. Upward deviation of both eyes deficient; defect more obvious in left eyes. No variation in tone or motor power. Knee-jerks on right side greater than on left, and right abdominal reflexes slightly less than left.

The diagnosis probably lies between "polio-encephalitis superior" and an initial stage of epidemic encephalitis.

(?) Cerebral Vascular Lesions: Case for Diagnosis.

By NEILL HOBHOUSE, M.D.

A. B., MALE, aged 56.

Onset in February, 1927, tingling and weakness in fingers of left hand; lasted one month and cleared up completely.

In October, 1927, sudden loss of feeling and weakness in right hand and foot. A month later had difficulty in walking. No sphincter loss.

The right hand is clumsy; no gross incoördination. Reflexes: abdominal, faint; knee-jerks, brisk left, sluggish right; ankle-jerks, brisk left, very sluggish right; plantars, flexor. No definite sensory loss, but appreciation is more acute in left hand than in right.

Systolic blood-pressure: 150 mm.; arteries tortuous. Cerebro-spinal fluid: Wassermann reaction negative. No cells. Globulin: no excess. Lange: no change.

The probable diagnosis is probably "multiple cerebral vascular lesions."

(?) Atypical Abiotrophy: Case for Diagnosis.

By NEILL HOBHOUSE, M.D.

MALE, aged 16. Six other children in family, all normal. Father "nervous." No history of any similar conditions.

Premature child (seven months). Confinement normal. Nothing abnormal noticed during infancy. Said a few words at age of 2 years. Did not walk until aged 4. When 8 years old was said to be deaf and dumb; went to school at that age; has never talked distinctly.

Vision is good, hearing fairly good. There is a severe degree of dysarthria; the boy dislikes trying to talk but can pronounce all consonants. He is able to read and write, and draws well. His mental development is better than his appearance suggests.

Physical Signs.—Irregular nystagmoid movements. Slight incoördination and clumsiness of hands. Reflexes: abdominals brisk; knee-jerks brisk; ankle-jerks present; plantars extensor. High degree of pes cavus in both feet. Gait: unsteady, with wide base. Marked lordosis.

Cerebro-spinal fluid: Cells 1. Protein 0·08 per cent. Globulin in slight excess. Wassermann reaction negative. Lange 1222100000.

The diagnosis appears to lie between a residual birth lesion and an abiotrophy allied to Friedreich's ataxia.

Delayed Cerebellar Atrophy.

By MACDONALD CRITCHLEY, M.D.

H. T. L., MALE, aged 67, has been attending the out-patient department for some six years (Dr. C. P. Symonds) on account of general unsteadiness. Symptoms commenced at the age of 55 with tremor of the hands.

In 1923 he showed nystagmus on lateral ocular deviation; bilateral intention tremor; ataxic gait; no abnormality of the reflexes. Wassermann reaction negative.

His condition has steadily deteriorated since then; in 1925, speech disturbance was first noted. At present he still has gross extension tremor and ataxia; he walks on a wide base and reels, especially on turning; the arms swing in an exaggerated manner.

Radial arteries hard and tortuous.

Blood-pressure 210/90.

The family history is entirely negative.

A Case of Thrombosis of the Lateral Artery to the Bulb.

By E. A. B. PRITCHARD, M.B.

THE patient, a previously healthy man aged 57, was suddenly afflicted four months ago with giddiness and ataxy of his legs while carrying straw to his sheep through the snow. He remained lying in the snow for two hours before he was found.

He has remained giddy ever since, especially on extending his neck.

For the past six weeks only he has noticed numbness and a feeling of cold over the left side of his face and the dorsal surface of his right hand. He has for the

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same period noticed that a cold object simultaneously applied to both legs (e.g. a water-closet seat) feels hot to his right leg.

On examination: no nystagmus; impaired sensibility to pin-prick and temperature difference over the cutaneous distribution of the left cranial fifth nerve —the dorsal surface of the right hand up to the wrist, and the right leg and side of the trunk up to the costal margin.

There is ataxy of voluntary movements with the left arm and leg without alteration of tone.

The point of interest is the distribution of the sensory impairment.

Section of Orthopædics.

President—Mr. W. Rowley Bristow, F.R.C.S.

[*May* 1, 1928.]

Case of Congenital Dislocation of Patella.

By W. Rowley Bristow (President).

I. S., A GIRL, aged 6, with bilateral congenital dislocation of the patellæ. The mother says that the child has always walked in a strange way. The patellæ are situated on the outer side of the knee-joints. There is an abnormal degree of mobility of the knee-joints in all directions, especially rotation in flexion of the tibia upon the femur. When the legs are extended the tibia is externally rotated upon the femur, and there is a separation of 4 in. between the malleoli. She is able to raise the legs in extension from the bed. She walks fairly well, but keeps her knees extended when doing so, and falls down if she attempts to run. She has weakness of the evertor muscles of the foot.

What treatment do Members advise, and when should treatment be undertaken?

We feel that we ought to deal with her at her present age, because there is no object in allowing her to grow up with the deformity. Our feeling is to transplant the tubercle of the tibia to the inner side, and split the capsule on the outer side, so as to allow the patella to be brought over. We are uncertain whether we should do a primary correction of the eversion of the tibia by a tibial osteotomy, at the same time correcting the degree of knock-knee which is present in spite of the laxity of the knee-joint.

In Paris we saw a case demonstrated by Dr. Mouchet in which he had brought the patella with the strip of quadriceps extensor attached to it through a button-hole in the capsule, and we propose to adopt this procedure.

Discussion.—Mr. Naughton Dunn said he considered that the rotation of the tibia was more apparent than real. Another interesting feature was that one could not obtain a forward position of the patella manually when the knee was flexed. He had seen a case in which, when that condition was present, the tensor femoris was inserted directly into the lateral border of the patella, and until that was divided, any form of treatment otherwise would be useless. He thought that the operation which Dr. Mouchet described and demonstrated in Paris would help in maintaining the position of the patella. He agreed with Mr. Bristow that the tubercles of the patella should be removed after the patella had been freed and centralized.

Mr. C. Lambrinudi said in cases like this he divided the capsule on the outer side of the patella and transplanted the semitendinosus and the sartorius into it. The result was good, and he thought that, on the whole, he preferred that method to the process of transplanting the tubercle. This patient might well be operated upon at her present age, because most of the pull on the sartorius was on the outer side, very little pull on the inner side contributed to the rotatory movement. He considered that reefing the capsule, and doing osteotomy later, would be enough. An osteotomy would, in his view, help to cure the condition.

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Mr. B. WHITCHURCH HOWELL said he felt that the rotation of the leg, especially on the right side, was due to an abnormal mobility of the knee-joint, and therefore could be ignored in favour of the treatment for the dislocation of the patella. Since the deformity was greater on the left side, and the skiagram proved that there existed a deformity of the femur, the internal condyle being larger, he thought that a cuneiform or a transverse osteotomy should be done, in addition to freeing the patella on the inner side. Then, with the re-education of the quadriceps, there would be no need to free the tubercle of the tibia and alter the insertion towards the inner side, especially as, in a child of this age—6 years—the tubercle was so minute. He agreed that operation should be undertaken at this age, and it should be done on both sides.

A Case of Dislocation of the Shoulder.

By B. WHITCHURCH HOWELL, F.R.C.S.

T. D., MALE, aged $15\frac{1}{2}$.

Complaint.—Deformity of right shoulder from birth.

Present Condition.—Post-glenoid subluxation of humerus. Forward flexion of humerus thirty degrees; internal rotation of humerus ninety degrees. Weakness of deltoid. No paralysis. Limb held in "hips firm" position, with hand palm

FIG. 1.

FIG. 2.

outwards. Patient cannot touch nape of neck with fingers. Skiagrams confirm clinical findings.

Proposed Treatment.—Excision of head of humerus to form an arthroplasty.

The interest of this case centres round a discussion whether it is a congenital subluxation, a dislocation of the shoulder-joint, or a residual deformity, due to Erb's paralysis, which was unnoticed at birth. The medical man who saw the child at

FIG. 3.

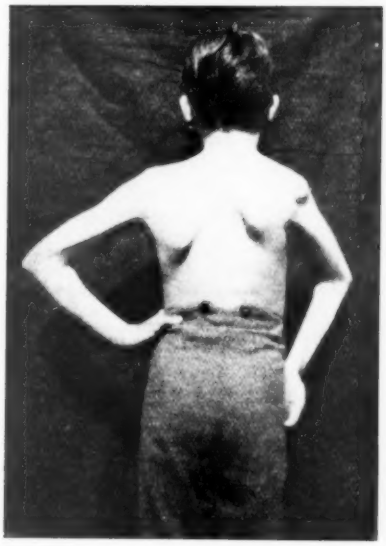

FIG. 4.

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its birth said there was no trauma, nor any sign of nerve involvement. I have read up the various cases of congenital dislocation of the shoulder-joint which have been recorded. Dr. Scudder, of Boston, U.S.A., was one of the first to describe it.

I cannot see how a shoulder, put in the position of dislocation and maintained there a sufficient length of time, can avoid the appearance seen in residual deformity of the shoulder-joint due to Erb's paralysis. The cases of congenital dislocation of the shoulder-joint which have been placed on record are very few in number—ten to fifteen, perhaps. Sir Frederick Eve described one in England. The photographs of Scudder's cases, and those of other surgeons in America, seem similar to those of this case.

With regard to treatment, I feel that to try to reduce the dislocation would be foolish, because the glenoid cavity is small and ill-formed, and the head of the humerus is malformed and is growing in the wrong direction. I do not consider that arthrodesis is necessary. He has got a good deltoid. He is unable to touch the nape of his neck with his fingers, and the elbow-joint is rotated through ninety degrees inwards. [See figures.] What I propose is excision of the head of the humerus in such a way and of such a length as to form an arthroplasty afterwards.

The condition is getting worse and I think something surgical should be done.

For the purposes of argument I suggest it is a congenital dislocation of the shoulder-joint.

Discussion.—Mr. ROCYN JONES said that if the case were really a birth palsy, then to produce such a complete and irreducible dislocation of the head of the humerus there would have to be a correspondingly severe paralysis of the shoulder girdle muscles. But if such a paralysis had occurred, there was now an astonishingly complete recovery of the muscles, which made him hesitate very much in believing that the arm ever had been paralysed. On the other hand, congenital dislocation would satisfactorily explain the limited, though free mobility, of the humerus, and its comparative stability, coupled with good muscle power. He therefore was inclined to agree with Mr. Whitchurch Howell in believing that the disability was a congenital one.

With regard to treatment, stable reduction of the dislocation was impossible; he therefore recommended an osteotomy of the humerus to correct the internal rotation and to improve abduction. This procedure was simple, devoid of risk and would certainly improve the function of the limb.

On the other hand, arthroplasty in this case would be a complicated operation, and was attended by a considerable risk of flail shoulder resulting, and he therefore would not recommend it. The boy already had a useful arm, and any further improvement would be best accomplished by osteotomy.

Mr. C. LAMBRINUDI said he considered that recovery had been too complete for the original trouble to have been Erb's paralysis. And the cases of Erb's paralysis he had seen had been, in nearly all instances, associated with stiff shoulders. He had never seen one with the degree of mobility possessed by this patient.

Mr. NAUGHTON DUNN considered the case an extraordinarily interesting one. The boy had fairly good movement in his shoulder-joint, and sufficient muscular power to elevate the arm well above the horizontal position and maintain it there. He (the speaker) agreed with Mr. Rocyn Jones that the internal rotation should be corrected, but it was

equally important that the power of getting the arm backwards should be established at the same time. He felt sure that the proper line of treatment at first was not only to correct the rotation after osteotomy, but also to get the arm well back. The boy could always bring it forward but would not be able to bring it back unless that possibility was established at the time of the operation.

Whether the condition was a congenital dislocation, or one following nerve trouble, he could not tell. He had not seen congenital dislocation of the shoulder, but he had seen a recurring dislocation of that joint due to laxity of the capsule. And he had seen a case in which there was a history of Erb's palsy, in which recovery had been equal to that shown by the present case.

Mr. ALAN TODD said the head of the bone was malformed, and there was no question of getting it usefully and painlessly in position. If one removed the head, there would be the difficulty of getting a stable, useful and painless shoulder. Surgeons did not nowadays seem to be so expert at excision of the shoulder-joint as in the days of such men as Sir Frederick Eve or Sir Henry Howse. He did not think excision of the head would give as useful a result for this patient as a good arthrodesis. With the latter one could confidently promise a painless, serviceable shoulder. If he had to deal with this case he would carry out arthrodesis.

Further, in connexion with his remark as to the skill of a former generation of surgeons in performing excision, there was to be seen in the Borough Market to-day a man whose two shoulder-joints had been excised by Sir Henry Howse. The man was a tuberculous subject, and Sir Henry excised both shoulder-joints and one elbow-joint. When he (the speaker) was Registrar at Guy's Hospital he had taken a photograph of this man holding out his hands in which were two fire-buckets full of water. He was still doing a full day's work in the Borough Market, which meant a life of great strenuousness. He (Mr. Todd) did not think many present-day surgeons could emulate such skill.

Mr. ROWLEY BRISTOW (President) said he saw no reason to go outside the ordinary diagnosis of Erb's paralysis in this case. The posterior and middle parts of the deltoid were extremely wasted and weak. He agreed with Mr. Dunn that many cases of Erb's paralysis had been seen which had recovered to the point that this patient had. The posterior subluxation of this boy's shoulder was extreme, but it was only a somewhat severe degree of the ordinary deformity. All other features presented by the patient satisfied, in his view, the diagnosis he suggested. The skiagrams showed that the whole arm on the affected side was smaller than its fellow; in fact he would have said that this was a typical case of Erb's paralysis, with a rather atypical degree of subluxation of the shoulder-joint.

With regard to treatment, he agreed with Mr. Dunn and Mr. Rocyn Jones in advising an osteotomy; if that failed to give good function, one could later arthrodese the shoulder.

Mr. B. WHITCHURCH HOWELL (in reply) said that for the sake of argument, he maintained that this might be a case of congenital dislocation of the shoulder-joint, and if one looked up the recorded cases bearing this title, Scudder's and others, the picture was rather like that presented by this boy, and also like a case of Erb's paralysis. On going minutely into the histories of the cases it would be difficult, as Scudder had found, to make a differential diagnosis. This boy had a general shortness of the whole limb, which Scudder said was a sign of congenital dislocation. He disagreed with Scudder because he (the speaker) considered the same condition was seen in Erb's paralysis.

With regard to arthroplasty or excision of the head to form a new joint, it was agreed, he thought, that the profession had now gone to the other extreme and were afraid to perform an excision of the joint. Most orthopædic surgeons, however, were now coming round to the

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belief that the future treatment of certain disabilities of joints would be an arthroplasty or an excision, rather than an arthrodesis; for example, in arthritis of the head of the femur. He had seen the case referred to by Mr. Alan Todd and he thought the reason of its success was that the surgeon had been bold enough to remove enough bone. If one were going to perform arthroplasty of the hip, one would not hesitate to remove the head, as it was known that the neck of the femur would not ankylose against the acetabulum. Where there was such a good deltoid and it was only atrophied in the posterior part owing to disuse, he saw no reason why treatment in abduction should not give a good result. Again, arthroplasty of the elbow-joint was a success, because, as a rule, there was a gap of an inch after removing the bone. Applying that to the case in question, it would be found that removal of the head of the humerus alone would not be enough. He was against scarring the axilla. Therefore removal of the head and sufficient of the shaft should give a good functional result.

He differed from the President in regard to atrophy of the deltoid; he thought the posterior fibres were stretched and that the condition was not due to paralysis, but to the pressure following subluxation backwards of the head of the humerus. It was not a question of paralysis due to involvement of the fifth and sixth cervical roots.

Section of Obstetrics and Gynæcology.

President—Mr. Comyns Berkeley, M.Ch.

[*May* 18, 1928.]

Perforation of the Uterus Associated with a Tubal Pregnancy.

Specimen shown by R. Christie Brown, M.S.

The specimen consists of the body of the uterus and right tube and ovary.

The right tube is the seat of an ectopic pregnancy, and the uterus shows two perforations, one at the fundus and the other low down on the left side.

The patient, aged 32, was admitted to the London Hospital on January 22, 1928. She was a married woman and had two children, one aged 6 years and the other aged 2 years. She had never had a miscarriage.

It was stated that she menstruated normally until November, 1927; with the November period, which commenced on the 25th, she had severe pain in the lower abdomen and bled profusely. Between November and January she menstruated every fortnight, and in the interval between the periods she suffered from an intermittent brown, watery discharge, occurring every four or five days. In between these periodic watery discharges, i.e., in the interval, she complained of a dull ache in the lower abdomen and felt generally ill. With the recurrence of the discharge the pain was relieved and she felt much better. Until the day of admission she had not consulted a doctor, but on this day the pain was so severe that she was compelled to go to bed and call in her medical attendant, who advised her to go to hospital.

On admission, temperature 99° F., pulse 104, patient pale and ill-looking. On examination by the abdomen there was a mid-line swelling reaching nearly up to the umbilicus; it was sub-resonant on percussion, and the upper part of the swelling felt cystic, whilst the lower part felt much firmer and of an entirely different consistence. Vaginal examination showed that the lower, firmer part of the tumour was continuous with the cervix and was thought to be the uterus. In the right fornix a harder, rounded mass, the size of an orange, was felt, and appeared to be fixed to the side of the uterus. It thus appeared that a bulky uterus was surmounted by a cystic swelling.

The patient was kept under observation for seven days.

On the second day her temperature rose to 100° F. in the evening, and she suffered from severe abdominal pain. During the night there was a copious brown, watery discharge. For the next three days her temperature remained normal and her pain was relieved. On the sixth day her temperature again rose to 100° F., the pain was more severe and again there was a watery discharge during the night. On the subsequent day, as before, her temperature was normal and the pain relieved.

I decided to examine the patient under an anæsthetic, whereupon the signs already stated were confirmed and it was found that the lower part of the mass was the uterus. A sound was passed and found to enter about 8 in. By feeling carefully on the abdomen the point of the sound could be felt peculiarly near to the abdominal wall. The withdrawal of the sound was followed by about one ounce of brown watery fluid. At this stage I thought I was dealing with a perforation of the uterus and decided to open the abdomen. On opening the

abdomen the anterior parietal peritoneum was found to form the anterior wall of an inflammatory cyst, which was opened up immediately the peritoneum was incised. The remainder of the cyst wall was made up of omentum, coils of bowel, uterus and adnexæ, matted together, the cyst itself being a localized accumulation of inflammatory exudate. The floor of the cyst was the fundus uteri, in which a perforation could be clearly seen. The cyst contained about one pint of a thin brown fluid. By gauze dissection the cyst was detached from the uterus and it was then seen that the right tube was distended and apparently filled with blood. This tubal swelling was the hard swelling which had been felt through the right fornix. I did a subtotal hysterectomy, conserving the left ovary, and in so doing I found another perforation in the uterus, low down on the left side, by means of which the uterine cavity communicated with the cyst cavity through a track which passed through the side of the uterus, the parametrium and the posterior peritoneal layer of the left broad ligament. The pouch of Douglas and the cavity of the cyst were drained and the patient made an uninterrupted recovery.

The patient was under the charge of Mr. Victor Lack, and I wish to express my indebtedness to him for allowing me to report the case.

A Case of Ovarian Pregnancy.

Specimen shown by J. Bright Banister, M.D., F.R.C.S., F.R.C.P.

Primary ovarian gestation is not very common and this specimen is a good example of the condition.

The patient, Mrs. H. J., aged 34, was admitted to the Chelsea Hospital for Women on February 18, 1928, as a case of right pyosalpinx. She stated that her last menstrual period was on January 8 and that three weeks ago she was seized with a sudden, acute pain in the lower abdomen, more marked on the right side. She fainted three or four times within the succeeding twelve hours. Ever since then, she had had dull, shooting pains in the lower abdomen and had vomited several times. The day before admission she began to lose a little blood from the vagina. Her obstetric history was uneventful. She had had four children all at full time and all normal deliveries. There had been no miscarriage. The menstrual history was normal, the periods recurring regularly every twenty-eight days.

On examination, she appeared a healthy woman. Abdominal examination revealed slight tenderness and rigidity low down in the right iliac fossa. Pelvic examination showed the cervix to be pointing downwards, not displaced to any degree forwards, and very tender on palpation. The body of the uterus was partially retroverted and there was a very tender and irregular mass filling the right side of the pelvis.

A tentative diagnosis of extra-uterine gestation was made and the abdomen was opened to find very dark blood in the peritoneal cavity, the right ovary enlarged with blood oozing therefrom, and apparently a normal right tube.

The right tube and ovary were removed and her further progress was uneventful.

The condition was regarded as either an ovarian pregnancy or a lutein cyst, into which hæmorrhage had occurred with a small leakage into the peritoneal cavity.

Macroscopically the specimen appears to be an ovarian pregnancy. The three criteria enunciated by Spiegelberg all appear to be present. The tube is apparently intact; the fœtal sac occupies the site of the ovary and is connected to the uterus by the ovarian ligament. Apparently too, ovarian structure is present in the sac wall (fig. 1).

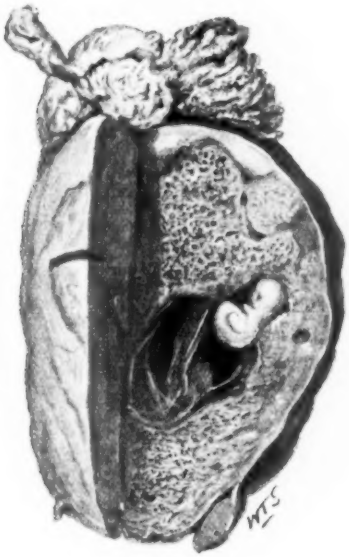

FIG. 1.

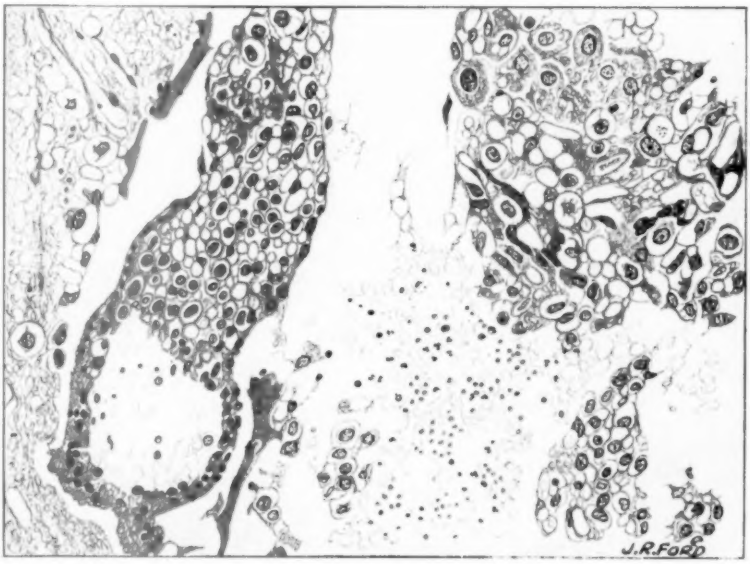

FIG. 2.

80 Gilliatt and Saunders: *Ectopic Pregnancy*

The microscopic slide is made from a section taken in the position demonstrated in the specimen. This slide shows blood-clot, obvious chorionic villi, and a capsule of connective tissue, in which there are several very large cells with large, well staining nuclei (fig. 2). These might possibly be either isolated cells derived from Langhans' layer, or interstitial cells of the ovary which have taken on a decidual reaction. Personally, I incline to the second explanation, and therefore I think that ovarian structure has been proved to be present in the sac wall.

Ectopic Pregnancy, Interstitial or Tubal.

Specimen shown by W. Gilliatt, M.S., F.R.C.S., and R. M. Saunders, M.B., Ch.B.

The specimen consists of the uterus, with the left ovary attached to it, and part of the left Fallopian tube remaining after a left salpingectomy performed in 1923.

The uterus has been opened transversely at the fundus to expose the uterine cavity and also a gestation sac, which may be either an interstitial pregnancy or one in the small portion of tube not removed at the time of operation.

The gestation sac has been cut across, and a piece of the wall of this sac taken for microscopic examination, the slide from which shows many chorionic villi lying amongst blood-clot.

Mrs. R. C., aged 33, first attended the out-patient department at King's College Hospital on April 17, 1928, complaining of continuous pain in the left side for four days, and a thick brown vaginal discharge. Her last menstrual period had been two weeks late but of normal character.

She had two children, the last nine years old; one miscarriage at the third month six years ago, and a left tubal pregnancy five years ago.

In 1923 the patient was admitted to St. Mary's Hospital suffering from an ectopic gestation and prolapsed ovary. Left salpingectomy and shortening of the left ovarian ligament were performed.

She was admitted to King's College Hospital the same day with a diagnosis of left ectopic pregnancy. The operation took place the following day, and when the abdomen was opened, the left tubal angle of the uterus was seen to be occupied by a dark swelling about 1½ in. in diameter, containing blood.

A panhysterectomy was performed, together with removal of the right tube and left ovary. The right tube and ovary were normal.

The interest of the specimen lies in the fact that in this patient, operated on five years ago for the condition, a left tubal pregnancy had again developed which, though it is difficult to determine the exact site, is either in the interstitial or isthmial portion of the tube.

A Case of Myelocytic Leukemia and Pregnancy.

By Everard Williams.

All reports on myelocytic leukemia and pregnancy emphasize the extreme rarity of the combination. Thus Kosmark writing in 1921 mentions twenty as the number of cases existing in the literature since 1888. He comments on twelve of these and remarks that the diagnosis has not been satisfactorily established in the remainder.

Chiari and Dantwitz writing in 1925 also reduce the number of properly authenticated cases to twelve, and they observe that of these twelve, only five resulted in the birth of a living child.

Hausam in 1922 discusses, among other points, the causation of the fœtal death-rate, but is unable to arrive at any definite conclusions.

Bower and Clark adopt the classification of myelocytic leukemia into acute and chronic cases. In the former the prognosis is so grave that it is doubtful if the condition of pregnancy affects the disease. There are apparently only two recorded cases, however, one by Bower and Clark, and an early one by Peterson. In both cases the disease developed during the course of an already existing pregnancy, and in one case proved fatal at the seventh obstetric month, and the other (Peterson's) one hour and a half after delivery, but he does not report whether at full term or not.

The only reported cases which throw any light on the influence of pregnancy on the chronic form of the disease are :—

Lambenberg's case in which there were four pregnancies, the last terminating by miscarriage at the fifth month, and followed in forty hours by the death of the patient.

Hausam's case in which the patient survived the birth of her child by five months ; and Chiari and Dantwitz's case in which the patient was known to be alive and well nine months after delivery, and was then lost sight of.

Bower and Clark's case (of chronic myelocytic leukemia) in which the patient survived delivery by only nine hours.

Present Case.—Mrs. C. H., aged 28, was in a medical ward of Charing Cross Hospital under treatment, by Dr Fenton, for myelocytic leukemia. She had had one confinement in September, 1926, but the child had died shortly after birth. She was suffering from abdominal pain and vomiting, a general feeling of illness, and a greatly enlarged abdomen. The symptoms had all been gradual in onset, and it was impossible to give a definite date to the beginning of the illness. The last menstrual period was on June 9, 1927, and since that date there had been two slight vaginal hæmorrhages.

On examination a mass having the characters of a splenic tumour reached almost to the pubes. The blood-count showed 174,000 white blood-corpuscles of which 32 per cent. were myelocytes. (For details of the blood-changes during the course of the disease see Table below.)

Date	Oct. 27	Nov. 12	Nov. 23	Dec. 6	Jan. 24	April 12
Red cells	4,400,000	3,900,000	3,900,000	4,300,000	4,300,000	2,900,000
White cells	175,000	160,000	222,300	128,000	206,000	283,000
Myelocytes	56,000	43,000	51,000	34,000	72,000	157,000

By way of treatment, in addition to small doses of arsenic, there had been X-ray exposures to the spleen, consisting of ¾ Sabouraud-pastille dose, unfiltered, from a 5-in. tube, on November 11 and 25, respectively.

I saw her on December 3, as there was some doubt as to the presence of a pregnancy, or, as to whether, if present, it should be allowed to continue. Examination showed a uterine tumour corresponding approximately in size with the period of amenorrhœa and having the characters of the gravid uterus.

In the absence of any evidence that termination of the pregnancy would have any beneficial effect on the course of the disease and the mother being anxious to have a child, it was decided to keep the patient under observation, and withhold interference until some definite indication should arise.

On January 6, 1928, i.e., in the thirtieth week of pregnancy, she was again admitted to hospital owing to a threatening miscarriage, but the symptoms subsided under treatment, and she was discharged on February 4. It was, however, decided to discontinue the X-ray treatment as it was doubtful whether the radiations could be excluded from the uterine tumour by any arrangement of screening.

On March 7 she was re-admitted as term had nearly been reached, and she lived some distance away from hospital.

On March 23 labour began during the night. The first and second stages were normal, and at mid-day the patient was delivered of a living female child by the mechanism of the first vertex. At 12.15 p.m. the placenta was spontaneously expelled with a good deal of hæmorrhage which was checked by hot douching, pituitrin and ergot. At 5 p.m. hæmorrhage started again, many clots were expelled, and she lost about a pint of blood. Blood transfusion was performed at 8.30 p.m. There was no further hæmorrhage until 2 o'clock the following afternoon when about a pint of blood was lost although the uterus felt firmly retracted. Two cubic centimetres of coagulin serum were injected, and there was no further hæmorrhage. The remainder of the puerperium was uneventful. A blood examination on April 12 showed 2,900,000 red cells and 280,000 white cells of which 157,000 were myelocytes.

The child did well, and a blood examination showed no evidence of any abnormal blood changes. Patient left hospital on April 14 with the uterus well involuted, still nursing her child, but in very weak health.

As one of the characteristics of the leukemias is the occurrence of hæmorrhages from the mucous membranes, or under the skin in the form of purpura, and as there is also an increase in the coagulation, and in the bleeding time, it would naturally be concluded that there would be a grave risk of death from hæmorrhage during parturition. However, reference to the literature shows that post-partum hæmorrhage is unusual, although the records of cases are not sufficiently complete to draw the conclusion that abnormal hæmorrhage has not occurred. In this case the hæmorrhage was sufficiently severe to necessitate a blood transfusion, and on the following day a further hæmorrhage occurred. However, the patient recovered and is still alive.

Some authorities consider that myelocytic leukemia is an abnormal response to an unknown infection, basing the hypothesis upon the known infectivity of myelocytic leukemia in fowls. If this hypothesis were correct, the infant might be expected to show the effects of the same infection, yet in all the cases in the literature in which the birth of a live child has resulted—five in number—the authors have laid stress upon the fact that the blood of the children, as in this case, was healthy.

The question arises as to how long the X-ray treatment for the leukemia should be continued during the pregnancy. One case, that of Chiari and Dantwitz, was treated by X-rays throughout the pregnancy and the puerperium. It resulted in the birth of a healthy child. In the other cases recorded, treatment was not continued throughout the pregnancy. In this case it was not considered advisable to continue the X-ray treatment after the twentieth week, owing to the possibility of injuring the fœtus.

My thanks are due to Dr. Fenton for allowing me to examine and record this case.

Through the kindness of Dr. Forsyth, physician to Charing Cross Hospital, I am able to include another case as yet unrecorded, which was under his care in hospital a few years ago. He has supplied me with the following details:—

"The patient was sent up to hospital by the doctor who had attended her in the confinement two or three days earlier, and on his first abdominal examination subsequent to the labour was astonished to find the abdomen largely filled by a solid tumour. This proved to be an enormously enlarged spleen, and the liver was also easily palpable. A blood examination showed the case to be one of myelocytic leukemia.

"I was interested to know what effect the mother's condition might have on the child, and I therefore arranged for the infant to be brought up for examination to hospital.

"It proved to be a full-time child, apparently healthy, and on physical examination no enlargement of the spleen or liver could be felt. Its blood, in striking contrast to that of the mother, showed no abnormality. In all other respects the child seemed perfectly healthy."

Report of a Sub-Committee Appointed by the Council of the Section of Obstetrics and Gynæcology to Prepare a Scheme for the Unification of the Clinical Reports of Maternity Hospitals.

THIS Sub-Committee was appointed in January, 1925, consisting of Dr. H. Russell Andrews, President of the Section, and Dr. T. Watts Eden, together with representatives of the lying-in hospitals of Great Britain and Ireland.

The Sub-Committee held several meetings at which representatives of several lying-in hospitals were present, while written communications were received from other lying-in hospitals whose representatives were unable to attend.

At the first Meeting, Mr. Rivett, representative of Queen Charlotte's Hospital, was elected Secretary.

The Sub-Committee considered the views expressed by the hospitals, either through their representatives or by letter, and it may be taken that the scheme upon which they finally decided is an agreed scheme which will be generally adopted, and will greatly increase the value of the hospital reports, since it will make it possible to compile aggregate statistics which include all the hospitals, and to compare the results obtained by hospitals situated in different parts of the country, and dealing with populations working under different industrial conditions.

A skeleton scheme has been prepared and is appended to this report. It is not intended to be exhaustive, or to indicate the manner in which all abnormal conditions should be reported.

The tables are presented as samples, and they can be expanded or compressed as may be required.

The Committee has drawn up the following scheme:—

1.—Patients admitted more than once during the same pregnancy whether for ante-natal treatment or on a false alarm of labour, count as one case only for all statistical purposes.

2.—All cases "booked by the hospital" for attendance at confinement, whether as "In-patients" or as "Extern District Patients," should be shown as separate sections of every table from those sent in, seriously ill or in labour with some complication, by outside doctors or midwives as "Emergencies." Cases advised to go to hospital to book for their confinement on account of some anticipated difficulty or complication, and which are accepted by the hospital, do not count as "Emergencies."

In the tables it is suggested that a division be made in each table after all the "Booked Cases" have been shown, followed by the details of the "Emergencies."

3.—The number of patients treated by the hospital should be given, showing how this number is made up, on the following lines:—

(A) *Patients "booked" in the Ante-Natal Department.*

(1) Delivered in Hospital.
(2) Admitted after delivery (born before arrival or from the Extern District).
(3) Discharged undelivered (after ante-natal treatment).
(4) Died undelivered.

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(B) *Patients sent in Seriously Ill, or with Some Complication, by Outside Doctors or Midwives, as Emergencies.*

(1) Before labour { (*a*) Delivered in Hospital. (*b*) Discharged undelivered. (*c*) Died.

(2) In labour ... { (*a*) Delivered in Hospital. (*b*) Died.

(3) After delivery.

(4) Abortions and ectopic pregnancies.

The total should be used to calculate all percentages and rates, but should be subdivided into "booked" and "emergency" cases.

4.—*A numerical summary* of all cases should be given, showing the number of the various presentations, normal deliveries and each type of abnormal case met with. This table should be in two sections: "booked" and "emergencies." It is obvious that it will contain reduplications; for instance, a breech presentation in a case of placenta prævia which develops pyrexia in the puerperium would be in the three categories.

(The Committee considered this to be one of the most important tables, as it shows the amount of abnormal work done at the hospital, and is valuable for determining the incidence of abnormalities. A specimen table is shown.)

5.—*Ante-natal Treatment.*—A table should be given showing:

(*a*) Number of cases.

(*b*) Condition for which admitted.

(*c*) Number delivered in the hospital.

(*d*) Number re-admitted for delivery later, or delivered by the hospital staff on the extern district.

(*e*) Number discharged undelivered for delivery by private doctor or midwife.

Cases admitted on a false alarm of the onset of labour should not be included in this table.

6.—*Vertex Presentations.*—The number of cases in which the occiput was anterior should be stated. Cases of posterior position of the occiput should be given in a table showing the treatment and termination (see specimen table).

7.—*Other Presentations.*

Breech presentations should be shown in tables divided into:

(i) Uncomplicated breech, which should be further divided into (*a*) Spontaneous delivery and (*b*) Assisted delivery.

(ii) Complicated breech (including primigravidæ) which should show complication, method of treatment, etc.

Face, Brow and Shoulder Presentations should be shown in tables giving cause (if any), treatment and results.

Prolapse of cord should be shown in a table giving complication or cause (if any), size of os when first diagnosed, treatment and results.

8.—*Contracted Pelvis.*—This table should include cases of relative disproportion between the pelvis and the fœtal head.

A summary of cases showing the number delivered by each method, with results, should be given.

Detail tables are good if space permits, but printing is more economical if they are shown in the tables of the different obstetric operations.

9.—*Ante partum Hæmorrhage.*—A summary of the different treatments and results in accidental hæmorrhage and placenta prævia should be given. Detail tables of each case are desirable if space permits.

10.—*Albuminuria and Eclampsia.*—A summary of the cases showing treatment and results should be given. Complete detail tables of each case are very desirable, and should show :

(*a*) Number of days in hospital before labour or discharge undelivered.

(*b*) Condition on admission, including amount of urine in first twenty-four hours, amount of albumin, œdema, etc., and blood-pressure.

(*c*) Condition on discharge, etc.

Albuminuria without toxic symptoms need not be given in detail and should be distinguished from albuminuria with toxic symptoms.

11.—Heart disease complicating pregnancy. The degree of cardiac failure should be stated in each case.

12.—Triplets, hydramnios, ectopic gestation, abortions, post-partum hæmorrhage and sundry other complications may be tabulated or dealt with in letterpress. Twins are usually well recorded and no comments were offered.

13.—*Obstetric Operations.*

Induction of Labour.—Detail tables showing the indication, method, length of time from induction to onset of labour, duration of first and second stage, weight of child, and such measurements of the pelvis and child as are recorded by the hospital.

Application of the Forceps.—Detail tables showing the duration of the first and second stage, weight of child and such measurements as are recorded.

Cases where the forceps have failed to effect delivery should be shown separately to the others in the "Booked" and "Emergency" tables, and the ultimate method of delivery stated.

Version.—Detail table showing the indication, type of version and result.

Cæsarean Section.—The usual details and whether the patient was in labour and, if so, for how long: whether membranes ruptured or not, and if previous attempts at delivery have been made.

Symphysiotomy and Pubiotomy.—Method and results and any details recorded.

Craniotomy and Embryotomy.—The indications should be given in each case.

Manual Removal of Placenta, Episiotomy and Repair of Perinæum.—May be given in detail or in the letterpress.

14.—*Morbidity.*

The Strassburg Convention standard of pyrexia, as adopted by the Ministry of Health, namely, "a temperature of 100·4° F. (38° C.) or more, sustained during a period of twenty-four hours or recurring during that period" should be used to calculate morbidity. These cases, with all deaths, to be given as a percentage of the total of cases treated as shown in the first table. Morbidity should be shown in a short table subdivided into "Booked" and "Emergencies."

15.—*Mortality.*

A full description of all maternal deaths should be given in the letterpress.

Reference to the mortality list and a footnote showing the rate for mothers and infants should be given in every table.

The mortality list should be subdivided into "Booked" and "Emergencies."

Note : Hospitals which admit cases of puerperal sepsis as such, after delivery by an outside doctor or midwife, should show them separately in both Morbidity and Mortality, and exclude them from the general rates.

16.—Full descriptions of unusual and interesting cases as given in the Queen Charlotte's, Rotunda and Coombe Reports are interesting and should be given if possible.

17.—Weights may be given in lbs. and ozs. or in grammes, preferably in both. If in only one, the same unit should be used throughout the report.

18.—Measurements may be given in inches or centimetres, preferably in both. If in only one, the same unit should be used throughout the report.

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Only such measurements as are recorded by the individual hospital are expected.

19.—Maturity or period of gestation should always be given in weeks.

20.—*Babies* should be shown under the headings :

(*L*) Living. (Alive on discharge.)

(*S B*) Stillborn (or dead birth), which is defined as failure to establish pulmonary respiration.

(*M*) Macerated (evidence that the fœtus died before labour commenced, or from some cause unconnected with labour).

(*D*) Died. (Born alive, but died before discharge from hospital.)

21.—*The feeding of the babies born at term* should be shown, together with the number that had gained and lost weight during their stay in hospital.

22.—*Premature babies* should be given in detail, giving birth, weight, method of feeding, cause of prematurity (if any), number of days in hospital and weight on discharge.

If only a summary is given the infants should be divided into groups by birth weights.

23.—*Fœtal Abnormalities.—Infants' diseases* and *infants' deaths* should be given as a list of causes and numbers.

We would repeat that the scheme is one of outline. Certain data should always be given in a comparable form, but the scheme is meant to leave scope for individuality, and the specimen tables are intended to illustrate the principles decided upon and are not the complete report.

Further, we would like to enter a plea here that all hospitals which issue these reports should circulate them among the other hospitals.

Maternity Hospital Report Tables.

NUMERICAL SUMMARY OF CASES DELIVERED IN HOSPITAL, ADMITTED FOR TREATMENT OR AFTER DELIVERY.

Presentations.

	Booked	Emergencies
Anterior Position of Vertex	—	—
Posterior	—	—
Breech	—	—
Face	—	—
Shoulder	—	—
Brow	—	—
Twins { Vertex / Breech / Transverse }	—	—
Contracted Pelvis including Disproportion	—	—
Ante-partum Hæmorrhage:		
(A) Accidental	—	—
(B) Placenta prævia	—	—
Post-partum hæmorrhage	—	—
Adherent Placenta	—	—
Albuminuria with Toxic Symptoms	—	—
Eclampsia...	—	—
Puerperal Pyrexia	—	—
B.B.A.	—	—
Intercurrent Diseases:		
Morbus cordis	—	—
Phthisis	—	—
Pneumonia	—	—
Ovarian Cyst...	—	—
Fibroids	—	—
Chorea gravidarum	—	—
Pyelitis	—	—
Encephalitis lethargica	—	—
Disseminated sclerosis	—	—
Neurasthenia	—	—
Exophthalmic goitre	—	—
Puerperal insanity, etc., etc.	—	—

Cases Treated in the Hospital Before Labour.

— Cases were treated in the Hospital before Labour, for the following conditions:—
— were discharged undelivered, one of whom returned later and was delivered in the Hospital,
the remaining — were delivered, with no maternal death.

Reg. No.	Age	Gravida	Maturity	Disease	Result	No. of days in Hospital before delivery or discharge	Remarks
Booked Cases.							
—	—	—	—	—	—	—	—
Emergencies.							
—	—	—	—	—	—	—	—

Note.—Details of cases of Albuminuria, Eclampsia and Heart Disease follow.

Vertex Presentation.

— cases of Vertex Presentation occurred.
The Occiput was Anterior in —, Posterior in —.
Treatment and Results of Posterior Position is shown below:—

Mode of Termination	No. of Cases	Results: Mothers L.	S.	D.	Children SB.	M.	D.	Remarks
Booked.								
Spontaneous Rotation	—	—	—	—	—	—	—	
Manual Rotation Forceps Delivery	—	—	—	—	—	—	—	
Spontaneous Delivery Face to Pubes	—	—	—	—	—	—	—	
Forceps Delivery Face to Pubes, etc.	—	—	—	—	—	—	—	
Emergencies.								
Manual Rotation Forceps Delivery	—	—	—	—	—	—	—	
Spontaneous Delivery Face to Pubes	—	—	—	—	—	—	—	
Forceps Delivery Face to Pubes ...	—	—	—	—	—	—	—	
Craniotomy, etc.	—	—	—	—	—	—	—	

— Mothers died, a mortality of — per cent.
— Babies died or were stillborn, a mortality of — per cent.

Contracted Pelvis.

Including Relative Disproportion between the pelvis and fœtal head.
— Cases were recorded during the year.
— Mothers died, a mortality of — per cent.
— Babies died or were stillborn, a mortality of — per cent.

Analysis of Cases and Results.

	No. of Cases	Deaths: Mother	Child	Mortality per cent.: Mother	Child	Remarks
Booked.						
Forceps (Labour not Induced) ...	—	—	—	—	—	
Induction (Spontaneous delivery)	—	—	—	—	—	
Induction (Forceps delivery) ...	—	—	—	—	—	
Induction (Cæsarean)	—	—	—	—	—	
Primary Cæsarean	—	—	—	—	—	
Craniotomy, etc.	—	—	—	—	—	
Emergencies.						
Forceps (Labour not Induced) ...	—	—	—	—	—	
Induction (Spontaneous delivery)	—	—	—	—	—	
Induction (Forceps delivery) ...	—	—	—	—	—	
Primary Cæsarean	—	—	—	—	—	
Craniotomy, etc.	—	—	—	—	—	

Details of these cases are shown in Tables of Obstetric Operations.

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Transverse Lie.

Transverse Lie occurred in — cases.

Reg. No.	Age	Gravida	Maturity	Complication	Treatment	Result M.	Result C.	Weight of child	Remarks
BOOKED.									
—	—	—	—	—	—	—	—	—	—
EMERGENCIES.									
—	—	—	—	—	—	—	—	—	—

— Mothers died, a mortality of — per cent.
— Babies died or were stillborn, a mortality of — per cent.

Prolapse of Cord.

Prolapse of the Cord occurred — times.

Reg. No.	Age	Gravida	Maturity	Size of os when diagnosed	Treatment	Result M.	Result C.	Remarks
BOOKED.								
—	—	—	—	—	—	—	—	—
EMERGENCIES.								
—	—	—	—	—	—	—	—	—

— Mothers died, a mortality of — per cent.
— Babies died or were stillborn, a mortality of — per cent.

Accidental Hæmorrhage.

— Cases of Accidental Hæmorrhage were treated.

Methods of Treatment	Concealed	Revealed	Results Mother L.	Mother D.	Children L.	Children SB.	Children M.	Children D.	Remarks
Spontaneous Delivery ...	—	—	—	—	—	—	—	—	
Rupture of Membranes ...	—	—	—	—	—	—	—	—	
Plugging Vagina	—	—	—	—	—	—	—	—	
Pulling down Leg	—	—	—	—	—	—	—	—	
Combined Methods, etc. ...	—	—	—	—	—	—	—	—	

Accidental Ante-partum Hæmorrhage.

Details of these cases are shown in the following Table.

Reg. No.	Age	Gravida	Maturity	Condition on admission	Albumin	Treatment	Result M.	Result C.	Remarks
BOOKED.									
—	—	—	—	...	—	—	—	—	
EMERGENCIES.									
—	—	—	—	—	...	—	—	—	—	—

— Mothers died, a mortality of — per cent.
— Babies were stillborn or died, a mortality of — per cent.

Cases of Albuminuria.

The urine of every patient is tested as a routine in the Ante-Natal Department, and in the Labour Ward when each patient is admitted. The routine treatment adopted was rest, strict dieting, alkalies and aperients. Labour was induced if the symptoms did not subside.
— Cases of Albuminuria were admitted for treatment and details are given in the following Table.

Reg. No.	Age	Gravida	Maturity	History of Renal Disease	Albuminuria on admission	Albuminuria on discharge	Œdema	Headache	Eye Signs	Highest Blood-pressure	No. of days in Hospital before labour or discharge	Type of Labour	Result Mother	Result Child	Remarks.
Booked															
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Emergencies															
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

— Mothers died, a mortality of — per cent — Babies died or were stillborn, a mortality of — per cent.

Eclampsia.

— Cases of Eclampsia were admitted during the year.

Reg. No.	Age	Gravida	Maturity	Condition on admission (including if in labour)	No. before admission	Fits Total	Fits Onset	Urine Albumin on admission	Urine Albumin on discharge	Quantity in first 24 hrs.	Œdema	Highest Blood Pressure	Headache	Eye Signs	No. of days in Hospital before delivery	Type of labour	Result M.	Result C.	Remarks.
Booked																			
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Emergencies																			
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

— Mothers died, a mortality of — per cent. — Infants died or were stillborn, a mortality of — per cent.

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Induction of Labour.

Induction of Labour was undertaken — times.

No. of Cases	Method		Indication	Result: Mother L.	Mother D.	Children L.	Children SB.	Children M.	Children D.
—	Bougies	—	Disproportion...	—	—	—	—	—	—
	Drug and Bougies	—	,, ...						
	Drug	—	,, ...						
	Stomach Tube ...	—	,, ...						
	Q.C.H. Bag ...	—	,, ...						
	Rupture Membs.	—	,, ...						
	Plugging Vagina	—	,, ...						
—	Bougies	—	Post Maturity	—	—	—	—	—	—
	Drug and Bougies	—	,, ,, ...						
	Drug	—	,, ,, ...						
—	Bougies	—	Albuminuria	—	—	—	—	—	—
	Drug and Bougies	—	,, ...						
	Drug	—	,, ...						
	Q.C.H. Bag ...	—	,, ...						
—	Bougies	—	Cardiac	—	—	—	—	—	—
—	Bougies	—	Intra-uterine Fœtal Death	—	—	—	—	—	—
—	Q.C.H. Bag ...	—	Eclampsia	—	—	—	—	—	—
—	Bougies	—	Pyelitis...	—	—	—	—	—	—
—	Bougies	—	Hydrammios	—	—	—	—	—	—
	Q.C.H. Bag ...	—	A.P.H.	—	—	—	—	—	—
—	Bougies	—	2 Extended Breech	—	—	—	—	—	...
—	Drug	—	2 ,, ,, ...						
—	Bougies	—	Static Œdema Legs	—	—	—	—	—	—
—	Q.C.H. Bag ...	—	Placenta Prævia	—	—	—	—	—	
—	Q.C.H. Bag ...	—	Premature Rupture Membranes ...	—	—	—	—	—	—
—	Bougies	—	Weak Uterine Scar. "Myomectomy"	—	—	—	—	—	—
—	Etc.	—	Etc.	—	—	—	—	—	—
Total —			Totals	—	—	—	—	—	—

Note.—Twins occurred — times.

Induction of Labour (Normal Delivery).

— Cases.

Reg. No.	Age	Gravida	Maturity	Indication	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	Duration of Labour 1st St.	Duration of Labour 2nd St.	Weight lb. oz.	Child Length	Circum. of Head	Result M. C.	Method	Remarks
Booked																
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—
Emergencies																
—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—

— Mothers died, a mortality of — per cent. — Babies died or were stillborn, a mortality of — per cent.

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FORCEPS.

Forceps were applied — times.

No. of Cases	Indication	Result: Mother L.	Mother D.	Children L.	Children SB.	Children M.	Children D.	Remarks
—	Prolonged Second Stage	—	—	—	—	—	—	
—	Fœtal Distress	—	—	—	—	—	—	
—	Pesistent Occipito-Posterior ...	—	—	—	—	—	—	
—	Maternal Distress	—	—	—	—	—	—	
—	Disproportion	—	—	—	—	—	—	
—	Cardiac	—	—	—	—	—	—	
—	Eclampsia	—	—	—	—	—	—	
—	Uterine Inertia	—	—	—	—	—	—	
—	Brow	—	—	—	—	—	—	
—	Prolapse Cord	—	—	—	—	—	—	
—	Fibroids	—	—	—	—	—	—	
—	Weak Scar. Myomectomy	—	—	—	—	—	—	
—	Not stated	—	—	—	—	—	—	
—	Etc.	—	—	—	—	—	—	
Total —								

There were — maternal deaths, a mortality of — per cent.
— Babies died or were stillborn, a mortality of — per cent.
In — Cases the forceps were applied after induction of labour.

Reg. No.	Age	Gravida	Maturity	
BOOKED				
9	28	2	40	Contracted Pe
34	41	3	40	,,
67	36	3	38	,,
424	21	1	39	,,
447	25	2	38	,,
643	23	1	40	,,
691	35	3	40	,,
996	39	1	40	,,
1201	26	2	40	,,
1257	28	2	40	,,
1259	28	1	40	,,
1334	24	2	40	,,
1541	24	1	40	,,
1692	35	2	40	,,
1739	34	2	40	,,
1084	39	3	40	Disproportion
398	37	7	40	Placenta Præv
1565	39	10	30	,, ,,
785	23	3	40	Obstructed La
1013	24	1	39	Parovarian Cy
1333	42	2	40	Age—Breech I
1335	28	4	40	Breech Presen
485	27	1	40	Cardiac ...
1388	32	3	36	Acute Intestin
EMERGENCIES				
59	34	1	40	Contracted Pel
301	21	3	40	Contracted Pel
494	27	1	40	,,
645	38	5	40	,,
790	39	1	40	,,
430	41	2	40	Disproportion
1877	24	1	40	Disproportion
1374	20	1	40	Obstructed Lal
557	44	5	36	Placenta Præv
920	42	2	38	Central Placen
937	28	1	36	Marginal Place
1072	37	2	40	Central Placen
1805	42	7	40	Central Placen
1003	38	7	26	Cardiac ...
628	22	1	12	Chronic Nephr
1617	30	2	32	Carcinoma Rec

Cæsarean Section.

Cæsarean Section was performed — times.

No. Cases	Indication	Result: Mother L.	Mother D.	Children L.	Children SB.	Children M.	Children D.	Remarks
—	Contracted Pelvis and Disproportion ...	—	—	—	—	—	—	
—	Placenta Prævia	—	—	—	—	—	—	
—	Cardiac...	—	—	—	—	—	—	
—	Breech. Previous Obstetric History, etc.	—	—	—	—	—	—	
—	Acute Intestinal Obstruction	—	—	—	—	—	—	
—	Carcinoma Rectum	—	—	—	—	—	—	
—	Ovarian Cyst	—	—	—	—	—	—	
—	Prolapse of Cord and Disproportion ...	—	—	—	—	—	—	

Cæsarean Hysterectomy was performed once for Chronic Neuritis—at third month.

Table of Case Details.

Indication	Int. Spin.	Int. Crist.	Ext. Conj.	D.C.	Duration of Labour 1st St.	Duration of Labour 2nd. St.	Weight lb.	Weight oz.	Child Length	Child Circum. of Head	Result. M.	Result. C	Remarks.
ed Pelvis	7¾	9	7¼	—	—	—	6	4	—	—	G.	G.	
,, ...	8½	9¼	7½	—	—	—	7	0	—	—	G.	G.	
,, ...	10	10½	7¾	—	—	—	6	3	—	—	D.	G.	Following Induction. See Maternal Death.
,, ...	8½	9½	6¾	4½	—	—	5	13	19½	13	G.	G.	
,, ...	—	—	—	—	—	—	5	14	—	—	G.	D.	Previous Embryotomy.
,, ...	8	9	6½	P.F.	—	—	6	3	—	—	G.	G.	
,, ...	9	10	6¾	P.F.	—	—	6	1	20	—	D.	G.	See Maternal Death.
,, ...	8	9¼	6½	P.F.	—	—	7	2	20	14	G.	G.	
,, ...	9¾	10¾	7½	P N.F.	—	—	7	8	—	—	G.	G.	1st Induction S.B.
,, ...	7¾	8¾	7	4	—	—	5	6	—	—	G.	G.	
,, ...	8¾	9¼	7½	3½	—	—	7	9	—	—	G.	G.	
,, ...	9½	10	6¾	P.F.	—	—	7	15	—	—	G.	G.	Previous Induction.
,, ...	10¾	11¾	6¾	4¼	—	—	7	3	20	14	G.	G.	Flat Pelvis.
,, ...	8	9½	7½	3½	—	—	6	10	—	—	G.	G.	1. Difficult Forceps. S.B.
,, ...	8¼	10	7¼	3¾	5	0	6	6	—	—	G.	G.	1 Craniotomy. Membranes Intact.
,, ...	9	10½	7½	P.N.F.	—	—	8	2	—	—	G.	G.	1 Craniotomy. 2 Stillbirth.
Prævia Centralis	—	—	—	—	—	—	8	1	22	14	F.	G.	See Morbidity.
,, ,, ...	—	—	—	—	—	—	3	6	—	—	G.	D.	
ed Labour ,, ...	—	—	—	—	—	—	8	14	20½	14	G.	G.	Failed Forceps "from District."
an Cyst	—	—	—	—	—	—	5	5	—	—	G.	G.	L. Broad Ligament Cyst.
eech Presentation	9½	10¼	8¼	P.N.F.	—	—	8	4	—	—	G.	G.	1st Baby neo-natal death. Anxious for living child.
Presentation and History ...	8¾	10½	7	P.F.	—	—	5	14	—	—	D.	SB.	3 previous Stillbirths. Craniotomy, Induction. Forceps. See Maternal Death.
... ...	9	9¾	7¼	P.N.F.	41	50	6	5	—	—	D.	G.	See Maternal Death. See Induction.
testinal Obstruction ...	8½	9½	6½	—	—	—	5	10	—	—	D.	SB.	See Maternal Death.
ed Pelvis (Obstructed labour)	—	—	—	—	3 days	—	7	4	—	—	G.	G.	In labour 3 days.
ed Pelvis	8½	10	6½	P.F.	—	—	6	3½	20	13½	G.	G.	Two previous Cæsarean Sections. Sterilization performed.
,, ...	—	—	—	—	—	—	5	11	—	—	G.	G.	
,, ...	—	—	—	—	—	—	7	13	21½	—	G.	G.	Bad Obstetric History.
,, ...	11	11½	6	P.N.F.	—	—	7	7	—	—	G.	G.	
rtion	—	—	—	3¾	—	—	7	1	21	13½	G.	G.	Previous Stillbirth. Anxious for living child.
rtion & Prolapse of Cord ...	—	—	—	—	13 hours	—	7	0	20	14	G.	G.	
ed Labour...	9½	10½	6½	—	16 hours	—	8	8	22	14	G.	G.	Membranes ruptured 16 hours. Fully dilated on admission. Bandl's Ring present.
Prævia	—	—	—	—	—	—	5	4	—	—	G.	F.	
Placenta Prævia	—	—	—	—	3 hours	—	6	15	20	—	G.	D.	
Placenta Prævia ...	—	—	—	—	—	—	5	15	—	—	G.	G.	Flat Pelvis present. Membranes ruptured and vagina plugged previously.
Placenta Prævia	—	—	—	—	—	—	9	15	22	15	G.	G.	
Placenta Prævia	—	—	—	—	—	—	7	14	21	14	G.	G.	
... ...	—	—	—	—	—	—	—	—	—	—	G.	D.	
Nephritis	—	—	—	—	—	—	—	—	—	—	G.	—	Cæsarean Hysterotomy.
a Rectum...	—	—	—	—	—	—	4	12	19	—	G.	D.	Colotomy performed.

— Mothers died, a mortality of — per cent.
— Infants died or were stillborn, a mortality of — per cent.

Section of Surgery.

SUB-SECTION OF PROCTOLOGY.

President—Sir CHARLES GORDON-WATSON.

[*May* 9, 1928.]

DISCUSSION ON THE EARLY DIAGNOSIS OF CARCINOMA OF THE RECTUM AND COLON.

Sir WILLIAM I. DE C. WHEELER (Dublin).

EXPERIENCE in the early diagnosis of cancer of the colon and rectum is given to few surgeons; I have seldom seen a case in the very early stage.

For the purpose of this discussion I referred to the records of forty-seven consecutive cases and found that most had been seen in the later stages when the diagnosis could not be mistaken, but when radical treatment was difficult or impossible. There were twenty-four cases of cancer of the colon and twenty-three of the rectum. Owing to the local nature of the malignancy, radical colectomy was possible in many more cases than excision of the rectum. Complete excision was possible only in seven (30·4 per cent.) of the twenty-three cases. There were no deaths. The colon was removed in twelve (50 per cent.) of twenty-four cases by various methods, with one death. Twenty-eight cases were beyond radical operation and were treated by colostomy (twenty-four cases); jejunostomy (one case); short circuit (two cases); radium and deep X-rays used with colostomy (two cases); no treatment (one case).

On a review of the literature I found that many surgeons had recorded the same lamentable experience of late recognition. Large numbers of patients are seen for the first time with acute intestinal obstruction, severe colicky pains associated with constipation or diarrhœa, anæmia, loss of weight and lassitude, the passage of blood, mucus and pus, or with a combination of all these, indicating terminal and widespread pathology.

Painstaking statistics have been tabulated by many authorities; the figures from independent sources support one another, but of these I will only say that 70 per cent. of cases of cancer of the rectum tabulated by Kutner [1] in the Breslau Clinic, were inoperable on admission, and I believe that the figure is approximately the same in St. Mark's Hospital, London.

Of 50,389 cancer deaths, Burgess [2] states that 26,670 were due to cancer of the alimentary tract, excluding the buccal cavity. Cancer of the sigmoid segment comes next to that of the rectum in frequency (excluding the stomach)—ninety-one cases in a series of 171 colonic cancers recorded by Brown [3]. Here again, although the number of operable cases of sigmoid cancer compares very favourably with that of the rectum, the mortality is still high when the early stage is passed.

I am purposely drawing attention to the relative frequency, the lack of early recognition, the high late mortality of cancers of the rectum, recto-sigmoid and distal portions of the colon, chiefly because it is just in these positions that it should be possible, with the means at our disposal, to make the diagnosis at an early stage. The great majority of these growths can be seen and felt, and are in this sense external cancers, comparable with cancers of the breast. If they are detected in the early stages, surgery has a rich reward—50 per cent. five years' cure in cancer of the rectum (Mummery [4]), and over 60 per cent. in the case of the colon (Peck [5]). With modern technique, operative mortality is becoming negligible (5 per cent. in my own series), the end-results are all that can be desired. (Rankin and Scholl [6]

record an operative mortality of 17 per cent. in the left half and 12·5 per cent. in the right half of the colon.) Early cancer of the colon is so easily extirpated that it is a great misfortune to the community that the diagnosis is so consistently missed. According to Butlin [7] 55 per cent. of the growths remain localized until death. In these circumstances, it is profitable to investigate critically our diagnostic weapons. Kutner, Binkley, Mayo, Jones, Mummery, Quick and many others comment on the failure or neglect of early and thorough examination. The first significantly remarks that in only one in three suspicious cases was a rectal examination made.

Binkley [8] found in a series of 300 cases that an average of ten and a half months elapsed before a definite diagnosis was made.

A rectal examination is of course highly desirable in every abdominal case, but specialists in the surgery of the rectum and colon should make allowances if the busy general practitioner is misled by pronounced upper abdominal symptoms. The early history of a colonic growth is sometimes so vague that it may point equally to such diverse conditions as cholecystitis and displacement of the uterus. That peristalsis is induced normally in the colon by certain foods, and that the bowel may be coaxed to empty itself when the patient smokes a cigarette, has a bearing on the early diagnosis of neoplasms. In the presence of a growth, whether from reflex spasm of the pylorus or from colonic peristalsis, early symptoms of "dyspepsia" may be induced by taking food. Distension, perhaps occasional vomiting and vague general pains after a meal, may point to an upper and mask a lower abdominal lesion. Thus the most alert diagnostician may be deceived before thorough examination reveals the truth. I have seen cases several times X-rayed after a barium meal when the *causa causans* was within easy reach of the finger in the rectum.

Now, as of old, cases of cancer of the rectum escape notice owing to an off-hand diagnosis of piles, and although the youngest student has been warned from time immemorial, it is surprising to find in recent records how many patients are victimized by this inexcusable mistake.

Again, digital examination is often perfunctory and haphazard; the words "nothing per rectum" so often recorded on hospital history sheets often mean nothing at all. By careless investigation small nodular cancers will escape notice and ballooning of the rectum will pass unobserved. In one of my private cases a growth in the rectum was mistaken for the cervix, mild uterine trouble co-existed and was treated by curettage. Many months later colostomy was performed for inoperable cancer. Students might well be taught to examine the pelvis bimanually per rectum in the same systematic fashion as is carried out per vaginam by gynæcologists. Examination in the erect or squatting position, or bimanual examination with or without an anæsthetic, will reveal growths high up in the rectum, or will detect indirectly involvement of the sigmoid loop. Gravitation growths in Douglas's pouch arising from the stomach or other distant focus may occasionally be found.

Just as tumours of the rectum and recto-sigmoid can be readily felt, so tumours higher up in the sigmoid (the site of cancer next in frequency to the rectum) can be readily seen. The use of the sigmoidoscope is indicated in every suspicious case where thorough rectal examination has given a negative result. Practitioners have been sufficiently informed neither of its simplicity nor of how to overcome the small difficulties in its use.

There is evidence that many carcinomata of the colon originate in pre-existing papillomata, and the early use of the sigmoidoscope may reveal such neoplasms in the pre-cancerous stage. Dukes and Burgess support this view.

Dukes (*Brit. Journ. of Surg.*, vol. 13, p. 720) states that evidence that cancers of

the bowel develop in preceding simple adenomata is offered by recent experimental work on the production of tar cancer in mice. Tumours thus experimentally produced are identical in appearance with the simple adenomata which accompany a cancer of the bowel. Cancerous degeneration of these experimental simple tumours occurred usually between six and ten weeks after they were first noticed. After the cancer had developed the remaining simple tumours progressed no further.

In a case of resection of the sigmoid followed by death, Mayo and Hendricks [10] found at autopsy that there were three mucous polypi in the cæcum; one was carcinomatous and similar to the growth in the sigmoid.

In the early search for cancers of the distal colon, with a few elementary precautions and a little practice the sigmoidoscope becomes "an elongated finger with a human eye." The most important of the elementary precautions is the proper preparation of the patient. If the bowel is not thoroughly emptied and quiescent examination is impossible. On the previous morning a saline aperient should be administered and followed by an enema. Three hours before examination the bowel should be washed out with a large saline injection, and two hours later, as recommended by Hill [12], the lower bowel should be cleared by half a pint of water containing half a drachm of glycerine. This last injection should never exceed the amount stipulated. When the preparation is complete, one hour before examination, $\frac{1}{4}$ gr. of morphia, as a rule, should be administered. The preparation will vary in different hands, but it is best to have some definite plan. The best view will often be obtained after normal action of the bowels without any special preparation.

The instrument is easily inserted in the knee-chest position and the best view will, as a rule, be secured. The obturator is not removed until the level of the middle of the sacrum is reached. The four valves of Houston then come into view. An occasional insufflation of air allows the tube to be passed onwards by sight. At the promontory of the sacrum, just as the instrument reaches the level of the pelvic colon, most difficulty will be encountered. The sphincteric arrangement at this level sometimes has the appearance of an impassable barrier of mucous membrane, and nothing but a blind pocket can be seen. The edge of this barrier approximates to the opposite side of the bowel, but relaxation occurs after a short delay and the nose of the sigmoidoscope can be insinuated beyond. Occasionally the instrument must be withdrawn a short distance and the path found very gently after re-introduction of the obturator. Landsman [13] and many others draw attention to specific groups of cases under observation for a considerable time without diagnosis, when a sigmoidoscope examination would have shown at once that a visible portion of the intestine was obstructed by a malignant growth.

X-ray Examination.

No examination is complete without radiography after a barium meal and enema. Here a warning is necessary to the family doctor or general practitioner who first sees the patient. Radiologists are too often regarded as miracle workers, and their dicta as akin to the gospel. They are, however, the first to admit that their powers of interpretation in gastro-intestinal diagnosis are limited. It is a grave mistake to take the report of the radiological examination alone as settling once and for all, the presence or absence of a colonic growth. A growth-producing partial obstruction may not interfere with the passage of the barium enema. A stricture of the sigmoid may be obscured by an overhanging coil if a barium meal has already been given. Technical difficulties render filling defects invisible, and there are many pitfalls and unsurmountable difficulties with which I am not in a position to deal.

62 Wheeler: *Carcinoma of the Rectum and Colon*

Carmen [14] points out that the most important sign of cancer of the colon is a filling defect and next in importance is obstruction, especially since non-malignant stricture of the colon is exceedingly rare. (See also Hurst [15].) The inflow of the enema is sometimes markedly or completely obstructed when the passage of the meal is apparently hindered only slightly or not at all. This is probably due to a valve-like effect of the growth. Dilatation of the bowel above the stricture is sometimes noted. A filling-defect, due to carcinoma, is characterized by irregularity and by constancy upon all plates and at successive examinations. It persists after the administration of anti-spasmodics. The filling defect of carcinoma is imitated by a variety of conditions, enumerated by Carmen: (1) Gas. (2) Insufficient quantity of the enema. (3) Fæcal matter. (4) Spasm. (5) Extrinsic tumours. (6) Pressure on bony parts. (7) Adhesions. (8) Diverticulitis, tuberculosis and other lesions of the intestine. It is not uncommon to observe a large persistent filling defect in a definite area of the colon, simulating carcinoma, continuing for some hours, and disappearing when atropine is administered in full doses. Pannett [16] has pointed out that if the affected bowel lies with its axis in the direction of the X-rays its defect will not be seen in the barium shadow.

Scott [17], Drummond [18], Shires [19], Hurst [20], Williams [21] and others have emphasized the importance of X-ray investigation.

Palpable Growths.

When a growth is situated low in the rectum or recto-sigmoid or lower sigmoid, over 90 per cent. can be palpated by rectal or bimanual examination (ninety-seven in 108 cases—Yeomans). At a higher level on the left a fullness or swelling often indicates a collection of fæces above a stricture. The "tumour" disappears after a copious enema. Such disappearance should raise and not dispel suspicion. Diverticulitis must be borne in mind: the symptoms of appendicitis transferred to the left side are most often due to this condition. Tumours on the right of the hypertrophiccauliflower form are often readily felt. Just as the presence of fæces is apt to be misleading on the left, so recurrent attacks of inflammation arising in and around a cancer of the cæcum may lead to a diagnosis of appendicitis on the right. Actinomycosis must be considered as rare in this situation: it is difficult to diagnose until a sinus has formed and sulphur bodies have been demonstrated with the microscope. Hyperplastic tuberculous infiltration closely simulates cancer: a pulmonary focus may very rarely be the guide to its recognition, but according to Hurst, there is no method of arriving at a differential diagnosis. The size of a tumour in the colon bears no relation to its malignancy, hence the importance of discovery when palpating the abdomen; in the stomach, on the other hand, a plainly palpable malignant tumour may be regarded as a death warrant. In the hepatic and splenic flexures, palpation is often difficult, but considerable assistance is obtained by palpation in the erect stooping position. The surgeon stands behind with both hands encircling the patient, and in this manner searches the upper abdomen under the ribs. The muscles are well relaxed and malignant tumours, encouraged by inspiration, descend as a rule to a lower level. Most of my patients were fat; there was usually flatulence and general distension, so that palpation was unconvincing. Absence of tumour proves nothing. A tumour in the left segment of the colon was discovered in only 24 per cent. of the 171 cases published by Brown.

The possibility of two independent tumours in different parts of the colon must not be forgotten. In one case I found a cancer of the sigmoid flexure and a cancer of the transverse colon in the region of the hepatic flexure, apparently quite independent of one another; in each case resection was done.

It has been suggested that the lower growths are implantations from those on the proximal side, or it may be that multiple papillomata in different portions of the bowel have undergone malignant degeneration. Mayo and Hendricks (*Annals of Surgery*, March, 1926) refer to the production of two carcinomatous areas at the same time, at widely separated areas in the large bowel. Miller [22] says "in a practical sense we must recognize that cancer of the colon may occur as multiple primary lesions." One case is recorded of removal of the sigmoid for cancer, and nineteen years later a second growth was excised in or about the splenic flexure.

When cancer is present, a certain degree of immunity to the establishment of further independent growths has been experimentally proved in animals, nevertheless there is conclusive evidence that whatever the underlying cause of cancer, it may produce unconnected lesions in the same individual.

Biochemical Examination, Blood, etc.

Blood, mucus and pus in the stools are valuable—but often late—indications of a colonic or rectal growth. Their presence sometimes leads to difficulty in the clinical diagnosis between cancer of the right side of the colon and ulcerative colitis.

Surgeons follow with interest discussions on the methods of testing for occult blood, and the attendant fallacies. During the first routine examination, however, the presence of occult blood is made certain by a positive benzidine test. It requires two hundred red cells per cm., to obtain the reaction. It is exceptional to find occult blood by this test in patients not suffering from some definite gastro-intestinal lesion.

Some authorities think that bleeding is more frequent in cancer of the distal colon, owing to injury of the mucous membrane by hardened fæces, and that this may be absent when the cæcum is involved. Marked anæmia, however, is often produced in the cæcum by toxic absorption, without any discoverable loss of blood.

Recent writings on the toxins of peritonitis and intestinal obstruction suggest the probability that *Bacillus welchii* plays an important part in the pronounced toxæmia associated with growths in this situation.

Burgess however thinks that the "cauliflower" type of neoplasm, chiefly found in the cæcum or the right half of the colon, tends to ulcerate early and to lead to diarrhœa and the passage of mucus and blood. He states that in this type of growth blood may be lost in small amounts over a long period, and produce so high a degree of anæmia as to lead to an erroneous diagnosis of pernicious anæmia. The hæmoglobin may be reduced to as much as 20 to 30 per cent., but this is not—as in cancer of the stomach—a contra-indication to operation. When there is marked anæmia secondary to cancer of the stomach, it almost invariably points to wide metastasis or is a growth inoperable because of local infiltration. Ulceration, according to Rankin and Scholl (*Arch. of Surg.*, vol. vii, 1923), seems to have relatively little bearing on the anæmia. Profuse hæmorrhage from cancer of any portion of the colon is not common. Friedenwald and Rosenthal [23] say that a blood-sugar tolerance test may be useful. It is claimed that usually in carcinoma of the gastro-intestinal tract there is a characteristic curve of sugar tolerance which differs somewhat from that of carcinoma of other regions of the body. Absence or diminution of free HCl is the rule. The Wassermann test should not be omitted, but I have never seen a stricture due to syphilis.

Exploration.

This should only be undertaken when symptoms are suggestive and persist and when all other means of diagnosis have been exhausted.

Fischer [24] gives the following good advice: (1) Where the X-ray is negative, the test for occult blood repeatedly positive, a tumour is not palpable and the history

is suspicious, wait and repeat the X-ray examination after from four to six weeks. (2) Where the X-ray findings are not definitely negative and cannot be explained by repeated examinations, a tumour is not palpable, the occult blood test is positive and the history is suspicious, perform an exploratory laparotomy. (3) Where the X-ray findings are suspicious, a tumour is not palpable, the tests for occult blood are negative and the history is suspicious, repeat the X-ray examination and stool examinations after four weeks and if the findings are the same, perform an exploratory laparatomy.

Suspicious Cases.

I have hitherto assumed that the patient is "under suspicion" and undergoing skilled routine examination. Perhaps, however, the most important consideration is the signs and symptoms which first bring the patient to the doctor. All the authorities mentioned draw attention to a gradual change from normal to abnormal bowel action. At first the change is not necessarily persistent, temporary improvement may occur. Osler pointed out that early gastric cancer should be suspected in a person past forty who developed dyspeptic symptoms which persisted, but who had had previously a "cast-iron" or "ostrich" stomach. So in the case of the colon, the regularity of the bowels disappears and flatulence and abdominal distress remain after purgatives.

On the other hand, the large bowel can propel fæcal matter through the narrowest of openings without causing much distress. In one case, under my care, of a growth the size of an orange, grossly and microscopically involving the small gut as well as the sigmoid portion of the large intestine, the patient had no obstructive symptoms. The tumour was discovered by accident; operation was advised but the patient felt so well that he only returned for operation twelve months later. Bowel contents passed so easily that it was very difficult to keep a cæcostomy opening patent during three weeks' preliminary drainage.

Very careful-history taking will throw a searchlight on significant signs and symptoms which to many patients appear unworthy of attention, but which have persisted in an otherwise robust individual for as long as two or three years. The age of the patient is helpful but by no means decisive. In one of my cases of cancer of the rectum the patient was under 20. Two cases are mentioned by Binkley between the ages of 10 and 20, and twenty-six between 30 and 40.

Fowler [25] (*Surg., Gyn. and Obstet.*, vol. xliii, p. 73) deals with a series of 118 cases of cancer in patients under 26 years of age; in 18·7 per cent., these were in the large intestine. Cancer of the colon in my experience is much less common in the young than cancer of the stomach or rectum.

Distension in some degree is often noticed in the flanks; later, when the small gut is distended by back pressure, the loops lie transversely and the well-known ladder pattern is produced. Visible peristalsis in coils of distended intestine is positive proof of obstruction. Even when coils are not visible, palpation may reveal a definite hardening of the bowel during a peristaltic wave, followed by relaxation and softening. This palpable peristalsis is sometimes very pronounced.

The lower bowel resents the presence of foreign bodies, including new growths, and teasing tenesmus is the constant result. For example: if a pedunculated polypus is present, there is constant straining in an attempt to expel it; if a plug of vaselined gauze is introduced beyond the sphincter as a temporary expedient, tenesmus often renders its presence unendurable. This teasing tenesmus with the discharge of mucus is therefore a sure sign of the presence of a neoplasm or foreign body. The discharge becomes blood-stained and offensive as ulceration proceeds.

Thus, although there is no early sign pathognomonic of cancer of the rectum or colon, there is a conglomeration of factors which should arouse sufficient suspicion to pilot a patient to hospital for the thorough detailed investigation which should never be omitted.

Finally, there remains the patient who does not seek medical advice. Constipation is relieved by purgatives, diarrhœa checked by astringents; anæmia and lassitude are combated by tonics and change of air. He is doctored by himself, his friends, and the press. Such useless sacrifice of life can be best met by education of the public. The campaign for this purpose has been happily inaugurated. Whatever the academic disadvantages, the public must be warned to avoid the neglect which leads to self-destruction.

Conclusions.

(1) Painful peristalsis of the colon or pyloric spasm immediately after food in the presence of growth, may lead to the faulty diagnosis of an upper abdominal lesion.

(2) Cancers of the lower colon and rectum are external cancers from the diagnostic point of view.

(3) Digital and sigmoidoscopic examinations are too infrequently made.

(4) Cancers of the hepatic and splenic flexures are best palpated with the patient in the erect stooping position.

(5) Cancer patients develop a certain degree of immunity, but the possibility of more than one independent growth should be considered.

(6) Palpable peristalsis is a reliable sign of obstruction in the colon; teasing tenesmus is a constant symptom of rectal growths.

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Dr. CUTHBERT DUKES.

The most useful contribution that I can make to this discussion is to leave the problems of pathology concerning the origin of malignancy, and limit myself to answering the practical question: "What can a pathologist do to help a surgeon make up his mind whether or not his patient has a cancer of the bowel?" The answer is disappointingly brief—his only field of service to-day is in the microscopic examination of a fragment of the tumour removed for diagnosis. Other laboratory investigations offer no help in answering this pressing question. We know of no reliable way of recognizing the changes in the blood that must accompany cancer from its beginning. Dr. Fry's blood test and similar serological reactions are not delicate enough to detect cancer in its early stage with sufficient discrimination to be of practical value. A year or two ago I had hopes of an agglutination test carried out with a peculiar strain of *Bacillus coli*, which was found to be more easily agglutinated by the blood-serum of patients with cancer of the bowel than by controls. These distinctive serological changes are due, no doubt, to the formation

of agglutinins by the defensive organization of the body, in response to infection of the bowel wall by common intestinal bacteria. The test was useless for diagnosis, because only in the later stages of cancer was it positive, and even then the results were often equivocal. Similarly the alterations in the urine, which eventually accompany rectal cancer, arrive too late to be of value in diagnosis. Chemical and microscopical examination of the fæces have been explored in vain. Since there are so many other possible sources of blood in the stool, and since the prospect of finding a fragment of cancerous epithelium in the daily dejecta is as promising as the search for the proverbial needle in the haystack, I think the pathologist may more profitably spend his energies in other directions.

However, there is one aspect of our recent work at St. Mark's Hospital on the pathology of early malignant growth that may be worth while for the surgeon to bear in mind when using the sigmoidoscope. As Mr. Mummery has convincingly proved from his clinical experience, the "innocent adenoma" of the bowel is a frequent percursor of cancer, and I have demonstrated to this Sub-section preparations illustrating the onset of malignancy in adenomatous tumours, and have shown that an early bowel cancer is often accompanied by a cluster of sessile adenomata and patches of epithelial hyperplasia, scattered over the mucous membrane for several inches above and below the malignant growth. The existence of these scattered, but widespread, epithelial proliferations warns us against the misconception that the malignant disease is strictly limited to the area of the bowel which bears the cancer. Their recognition might be of use in diagnosis. If, for instance, on sigmoidoscopic examination no obviously malignant tumour were discovered, but several of these innocent looking adenomas were noticed in the rectum, this observation should determine a very close medical supervision in the immediate future, with frequent re-examination with the sigmoidoscope.

To return to microscopic examination of a fragment of a suspected cancer, it is worth while to consider which region of the tumour is most likely to permit a decisive opinion, and the best method of removing the fragment. These two questions are important, for a pathologist decides on the presence or absence of malignancy partly from the atypical appearances of the epithelium under high magnification, but chiefly from the general disposition of the cells and their relation to neighbouring tissues. Too much reliance should not be placed on the morphology of individual cells, for in a rapidly growing adenoma in which the natural relation of epithelium is strictly preserved and in which there is no encroachment into subjacent tissues, cells may be found which illustrate all the accepted distinguishing marks of malignancy, such as absence of secretion of mucus, hyperchromatic nuclei, and irregular mitotic figures. The individual cancer cell possesses no histological feature that may not be paralleled in a cell separated from a non-malignant tumour. With isolated cells, then, the highest magnification cannot distinguish the malignant from the benign. Much more can be learnt from the general arrangement of the epithelium, but this also is not an infallible guide, because the disorderly disposition of the cells in cancer may be closely mimicked by the proliferation of a benign tumour. The only unequivocal sign of cancer of the bowel is intrusion of atypical epithelium into regions where such epithelium is not normally found. This encroachment is incontrovertible evidence of malignancy. The failure to discover evidence of invasion in a piece of tissue large enough to provide an adequate field of view warrants the decision that the tumour is non-malignant.

The features which the pathologist wishes to study are most conspicuous in the tissue at the edge of the tumour or ulcer. A fragment removed from this region will show the normal epithelium side by side with the neoplastic, and allow of comparisons being made between the natural histology and the disorderly disposition of the new

growth. Moreover, the margin of a cancer is its growing point, and therefore a fragment from the edge is the best sample for the recognition of the nuclear peculiarities of malignant cells.

The removal of the fragment is almost as important as its site of selection. It should be handled carefully, not squeezed or crushed during removal, nor cut until properly hardened. Removal by dissection is the method of choice, but almost equally satisfactory samples can be obtained by means of a punch. The detached portion of the growth should be dropped into 10 per cent. formol saline or other fixative as soon as possible after removal.

With regard to the interpretation of the pathologist's report. If the surgeon has insisted on a frozen section in order to save time, he must be prepared, in some cases, for an initial non-committal report, and wait for a more definite finding until the four or five days necessary for preparing a paraffin section have elapsed. Although a frozen section prepared in a few hours is often good enough for a final report, in doubtful cases the clearer picture which the paraffin method provides may be needed for the ultimate decision. Finally, a negative report stating that no evidence of malignancy is discoverable does not necessarily imply that the whole tumour is benign. The report refers only to the portion sent for examination.

Dr. H. K. Graham Hodgson.

In considering the often apparently trivial symptoms of early carcinoma of the colon, it is not surprising that in many cases much valuable time is lost before the symptoms become sufficiently disquieting to induce the patient to seek medical advice.

For this reason the radiologist sees the early stages of a small filling defect much less commonly than the gross and more advanced changes in the bowel wall due to carcinoma.

Recognizing the extreme importance of the early recognition of carcinoma of the colon, how far can the radiologist assist the surgeon in his diagnosis?

An efficient X-ray examination can be of great assistance, by confirming the clinical suspicions, locating the growth, and defining its extent. The most important radiological sign of carcinoma of the colon is a filling defect due to the encroachment of the growth on the barium-filled lumen. The filling defect may be coincident with a palpable tumour. Other signs are spasm, obstruction to the flow of the enema, localized absence of peristalsis and distension of the bowel with gas proximal to the constriction.

A growth causing a gross and obvious filling defect in an easily viewed part of the bowel gives rise to no difficulty in its radiographic demonstration. But the smaller the filling defect, the more important and the more difficult is its detection, and, especially when an early growth is in a part of the colon where the calibre is wide, as in the cæcum, or where it is covered by the shadow of overlying loops, as in the sigmoid colon and in the hepatic and splenic flexures, is very apt to escape observation if screened and radiographed in the antero-posterior plane only. In these situations oblique views are essential.

It is not uncommon to see what appears to be dilatation of the rectum, with obstruction to the further progress of the enema. With the antero-posterior view the radiological inference is that a growth is present at the upper end of the rectum. An oblique screen examination, or radiogram, however, will frequently show that the growth is situated in the sigmoid colon, often some distance along it. The obstruction causes a ballooning of the sigmoid and rectum so as to show only a single shadow in the antero-posterior view, but the oblique view throws the shadows of the distended rectum and sigmoid clear of one another.

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Fig. 1 shows an apparently normal rectum and sigmoid colon in the antero-posterior position, except for this solitary diverticulum, but the oblique view (fig. 2) throws the whole sigmoid clear of the rectum and reveals this filling defect.

Such cases clearly demonstrate that it is impossible to exclude a filling defect unless every inch of the large bowel has been screened and radiographed so as to

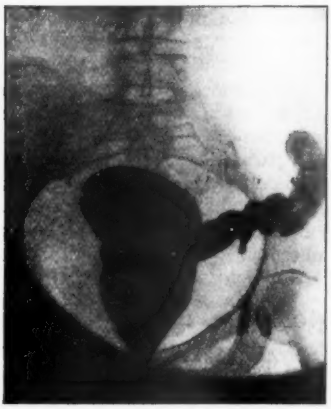

FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

throw it clear of the shadows of overlying loops, and this can only be done by making a routine use of oblique views.

Dual Exposures.—Another method of great use in detecting early malignant or inflammatory invasions of the bowel wall is the dual exposure. Two radiographic exposures are made at intervals of two or three seconds on the same film, the

patient ceasing respiration until both exposures have been made. The bowel being distended with warm fluid, peristaltic action is more frequent than normal. In the radiogram the outlines of two or more peristaltic waves can be seen in a healthy bowel. When, however, the bowel wall is diseased, only a single outline is seen through the diseased area, owing to the localized cessation of peristalsis (figs. 3 & 4).

Though carcinoma is the commonest cause of a filling defect in the shadow of the barium-filled colon, filling defects, both apparent and real, may be brought about by other conditions.

An apparent filling defect, due to localized spasm, has frequently to be distinguished from a true filling defect. Such a spasm may be caused by a lesion of the bowel wall, or from a pathological condition extrinsic to the bowel. On the distal side of a colonic growth the passage of a barium enema is frequently completely obstructed, for the time being, by a valve-like spasm. If the examination is discontinued at this point there is some doubt as to the cause of the spasm and the exact position of

FIG. 5.

FIG. 6.

such a cause. This difficulty can usually be overcome by decreasing the height of the enema container to 12 in. or so above the colon and maintaining this pressure constant for five or ten minutes, or even longer, whilst gently massaging the spastic area. After a while the spasm gradually relaxes and the barium fills the bowel beyond, so showing the cause of the spasm, if present in the colon.

The following three cases show spastic obstruction to the enema, due to different causes. In the first one (fig. 5), the barium is completely obstructed in the neighbourhood of the splenic flexure. After maintaining constant low pressure and massage for a few minutes, the spasm relaxed, showing a napkin-ring constriction in the distal part of the transverse colon (fig. 6).

The second case showed a similar spastic obstruction, but near the hepatic flexure. Again using the procedure described, the spasm relaxed and I found, beyond the site of the spasm, a normally filling colon.

A dual exposure radiogram showed normal peristaltic waves flowing through this part of the bowel, strongly suggesting that the bowel wall was healthy. After a few minutes the spasm returned but was diffused over about 6 in. or so of bowel.

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On the spasm again being relaxed by this method, I rotated the patient into the oblique position and saw a very faint shadow above the hepatic flexure, which disappeared as the patient was returned to the supine position (fig. 7). On this evidence I reported a large, solitary gall-stone, probably causing the spasm, with no X-ray evidence of disease of the bowel wall. At operation a large, flat, single gall-stone was found. The gall-bladder was not adherent and the colon was healthy.

FIG. 7.

FIG. 8.

The other case shows a chronic appendix causing a spasmodic constriction of the cæcum (fig. 8).

Another cause of pseudo filling defects is incomplete emptying of the colon prior to X-ray examination, which should never occur where adequate preparation is possible. Such a pseudo filling defect, on the radiographic evidence alone, might

suggest carcinoma. Palpation under the screen, however, usually reveals its true nature, but such cases show how necessary thorough preparation is.

True filling defects, however, can be very deceptive in their appearance. Fig. 9 shows a case with all the appearance of early colonic carcinoma. There was this constant filling defect in the distal transverse colon, and coincident with it was a

FIG. 9.

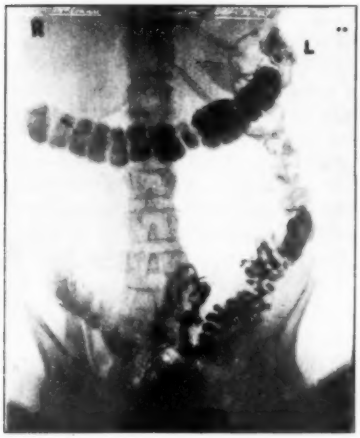

FIG. 10.

palpable tumour, fixation spasm, and localized absence of peristalsis, but the condition found at operation was fixation of the colon at this point by a mass of adhesions in the omentum, and no disease of the colon itself.

Even in cases of diverticulosis it is not invariably safe to assume that a narrowing of the bowel lumen is of inflammatory origin (fig. 10). In this case numerous

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diverticula are present, and it was possibly pardonable to presume that this filling defect, indicated by arrows, was inflammatory in origin. But it was, in fact, due to carcinoma.

Such cases show that, although the X-ray examination may demonstrate a filling defect, the interpretation as to the actual cause is the province of the clinician.

Finally I will mention a possible surgical complication which may occur in the event of operation following shortly after a barium enema examination. I have found two such cases—where a colotomy was performed a day or two after the X-ray examination. In both these cases obstruction supervened owing to a hard inspissated mass of barium becoming impacted in the bowel lumen proximal to the colotomy opening. Such a complication can be avoided by a precautionary screen examination prior to the operation.

I thoroughly agree with Sir William de Courcy Wheeler with regard to the value of negative X-ray evidence.

The earliest demonstrable X-ray sign of carcinoma of the colon is the filling defect, and prior to the growth attaining a sufficient size to cause a filling defect there will be no definite X-ray evidence indicative of invasion of the bowel wall, except that a dual exposure radiogram may show localized absence of peristaltic action. The generation of suspicion in the patient's mind is slow; these suspicions are extremely valuable as they lead to vigilance which will make possible operation at the earliest moment. The radiologist should, therefore, not unduly allay these valuable suspicions on negative X-ray evidence alone. A further examination in three weeks or a month may show a very different picture.

Therefore, though an X-ray examination is a very useful link in the chain of evidence, it was never intended to be, and cannot be, used as a sole means of diagnosis of carcinoma of the colon, but simply to elucidate and amplify those facts which cannot be definitely determined by the clinical examination.

Dr. Arthur F. Hurst.

The early diagnosis of carcinoma of the colon depends primarily upon the ability of the practitioner to obtain an accurate history of the patient and to recognize what symptoms should lead him to suspect that this disease is present. By the time the first symptoms appear a growth is likely to be of such a size that a definite diagnosis can be made by means of the X-rays, the sigmoidoscope and examination of the stools, or, in the case of the rectum, by simple digital examination.

Table I gives the duration of symptoms, at the time of admission into New Lodge Clinic for diagnostic purposes, of twenty-five consecutive cases of cancer of the colon and rectum.

Table I.—Duration of Symptoms in Twenty-five Cases sent for Diagnosis.

Number of cases	Duration of symptoms in months	Number of cases	Duration of symptoms in months
2	Two	5	Twelve
3	Three	2	Sixteen
1	Four	3	Eighteen
2	Five	1	Twenty-four
4	Six	1	Thirty-six
1	Eight		

The case in which symptoms had been present for three years was one in which malignant degeneration had occurred in a group of polypi in the descending colon. As an early diagnosis and operation might have prevented the development of carcinoma, there is no reason why it should not appear in the table. The average duration is ten and a half months or nine and a half months if the case of polyposis

is excluded. It is clear therefore that the prospects of radical cure by operation would have been very different if all the patients had been sent for investigation directly they complained of suspicious symptoms.

The two earliest symptoms are abdominal discomfort or pain and a change in the habitual action of the bowels. Both symptoms were present in almost all our cases. The discomfort or pain is localized in the segment of bowel proximal to the obstruction. It is a small intestine pain and is felt in the neighbourhood of the intestines in cancer of the cæcum. In cancer of the ascending colon or hepatic flexure it is felt on the right side of the abdomen, and in cancer of the splenic flexure, descending and iliac colon, and the proximal part of the pelvic colon, it is felt on the left side. Pain produced in the middle segment of the transverse colon is felt just below the umbilicus, and that caused by growths in the distal part of the pelvic colon or pelvi-rectal flexure are localized in the middle line between the umbilicus and pubes. The pain is often colicky, and its disappearance may coincide with a gurgle which can be heard and felt; this is a most significant symptom, which may enable the patient to localize the site of obstruction with great accuracy.

In nine out of twenty-five cases the patient had become more constipated and in six had complained of diarrhœa; in eight cases there was alternately constipation and diarrhœa, or an initial attack of diarrhœa followed by persistent constipation. It is necessary to add that in cancer of the rectum and pelvic colon the diarrhœa is of a spurious type, examination of the stools showing that solid fragments are mixed with the semi-liquid fæces. In only two cases had no change in the habitual action of the bowels been observed.

In contrast with carcinoma of the stomach general symptoms are rarely observed in the early stages. The patient may feel quite fit, with a good appetite and undiminished energy. No anæmia is present and no weight is lost. In only one of this series of cases and in one other earlier case were general symptoms present without intestinal symptoms. The patients were anæmic and complained of anorexia and loss of energy. As occult blood was found in their stools and complete achlorhydria was present, carcinoma of the stomach was diagnosed, though the X-rays had shown no abnormality. A growth of the ascending colon was, however, found in one case, and a growth of the splenic flexure in the other.

Radiological Diagnosis.

An opaque enema is of great value in demonstrating the site of any obstruction in the colon, as I first showed just twenty years ago. It is a curious fact that the enema may be temporarily or permanently held up by an obstruction, which leads to no obvious delay in the passage of an opaque meal. The passage of the fluid should always be watched, as a slight obstruction may only lead to a temporary delay in the passage of the fluid, caused possibly by spasm. The point where any delay is observed should be palpated under the screen, as it is not uncommon for a small tumour to become palpable when one's fingers are directed to its exact localization. Before accepting the apparent "hold-up" of an enema as evidence of a stricture, the patient should turn on to his face, as the fluid occasionally passes on in a normal manner when this is done.

A good radiogram may show the nature of the obstruction, as the narrowing caused by adhesions, diverticulitis and a fibrous stricture, following ulcerative colitis, all give appearances which are to some extent characteristic and differ from the irregular jagged outline of that produced by a growth. A filling defect is sometimes recognizable in the complete absence of any obstruction.

74 Hurst: *Carcinoma of the Rectum and Colon*

It is very important to remember that early cases of carcinoma of the colon may show no filling defect and cause no obstruction to the passage of an opaque meal or opaque enema. In 1909, and again in 1915, before I fully realized this, I rejected the diagnosis in two cases in which the symptoms had made me suspect a growth of the colon. Within three months a tumour became palpable in both cases; the delay in advising operation may have caused the recurrence.

As a definite diagnosis had been made by rectal and sigmoidoscopic examination in five out of the series of twenty-five cases, an opaque enema was given in only twenty. It showed obstruction in fourteen of them and a filling defect without obstruction in the others; in four of the former an opaque meal also showed obstruction. In one case the meal showed obstruction 4 in. above the level of the iliac crest, where the opaque enema had been obstructed; corresponding with this, the growth was found to have formed the head of an intussusception 4 in. long. In one case an opaque meal revealed an obstruction which was not shown by an enema, and in one case neither enema nor meal showed any abnormality.

The Stools.

Mucus with a solid stool is of no clinical significance, but blood or pus, with or without mucus, is always an indication that further investigations are required. If associated with entirely fluid or semi-fluid stools, ulcerative colitis is in all probability present, but if in addition, fragments of solid fæces are found, even if the patient complains of "diarrhœa," a growth of the pelvic colon or rectum is a more likely diagnosis; visible blood and pus, however, are hardly ever observed when the growth is proximal to the pelvic colon.

When no abnormal constituents are recognized with the naked eye, a farinaceous and chlorophyll-free diet should be given with some charcoal. As soon as all the charcoal has been passed the stools should be examined chemically and spectroscopically for occult blood. In our experience this is present in all, or almost all, specimens in every case of cancer of the stomach and of the colon. Obvious blood was present in the stools in four cases involving the rectum, and in one out of eight involving the pelvi-rectal flexure, but in only one other—the growth of the iliac colon which had formed a chronic intussusception. Occult blood was found in all the remaining nineteen cases. In each case a positive guaiac reaction was obtained; in four this finding was confirmed by an extract giving a spectrum of hæmatoporphyrin alone, in three of acid hæmatin alone, and in two of both hæmatoporphyrin and acid hæmatin. The acid hæmatin spectrum is of special importance, as it is rare in cancer of the stomach or ulcer of the stomach or duodenum except after a large hæmorrhage, and it is then always accompanied by the spectrum of hæmatoporphyrin. Of the nineteen stools containing no obvious blood, four had some excess of mucus, and in two of these and one additional case the patient had occasionally noticed traces of blood before admission to the clinic.

Occult blood is rare in uncomplicated diverticulitis and never occurs in constipation or diarrhœa unaccompanied by organic disease. Curiously enough, a local band of adhesions involving the colon may also lead to constant occult blood in the stools. As this condition causes symptoms and radiological signs indistinguishable from those caused by a growth, the differential diagnosis may be impossible, though there is generally a longer history of illness, the details of which often point to the probable cause of the adhesions. Fortunately it does not greatly matter if a mistake in diagnosis is made, because this type of adhesion is the one form which can generally be dealt with satisfactorily by operation.

Sigmoidoscopy.

I need say nothing here of the value of the sigmoidoscope in the early diagnosis of carcinoma of the pelvi-rectal flexure and pelvic colon, but I will suggest, as I have often done before, that it is time that the profession as a whole learnt to use this instrument which should not be regarded as only to be handled by the proctologist. Nobody thinks of diagnosing pharyngitis without direct inspection of the pharynx; nobody should think of diagnosing hæmorrhoids or colitis without digital examination of the rectum and direct inspection of the colon. If this rule was always followed, the lives of many patients, who now reach the surgeon too late for radical operation, would be saved. A patient in whose rectum I had found a large inoperable growth had seen no fewer than five medical men within three months of consulting me, not one of whom had made a rectal examination. I have also seen a number of patients with growths of the rectum or pelvic colon, who had just undergone a course of Plombières douches at a spa, in whom a digital or sigmoidoscopic examination would have at once revealed the nature of their disease. For successful sigmoidoscopy neither an anæsthetic nor morphia is required, and the patient should, if possible, be examined without any preparation by aperients or enemata.

In the present series of twenty-five consecutive cases a tumour was palpable on abdominal examination in six, in one of which it was only discovered whilst palpating a visualized filling defect under the screen during an X-ray examination. In all four cases of rectal carcinoma the tumour could be felt on ordinary digital examination, and in one case involving the pelvic colon it was felt through the anterior rectal wall. The pelvi-rectal flexure—the commonest site of carcinoma of the bowel—is $4\frac{1}{2}$ in. from the anus and has only a very small range of movement. A growth in this situation is therefore beyond the reach of a finger inserted *per anum* and is rarely palpable on abdominal examination, but it can easily be recognized with the sigmoidoscope, as it was in the eight cases in our series. In seven cases the tumour was beyond the reach of the sigmoidoscope, and was also so small, or, in the case of the splenic flexure, so high up under the left costal margin, that it could not be felt on abdominal examination.

If all cases of suspected carcinoma of the colon were sent for diagnosis within a month of the onset of symptoms, many patients could be advised to undergo an operation when the evidence was still inconclusive. But an occasional unnecessary exploration will be more than compensated for by the infinitely better prospects of permanent cure, if the average duration of symptoms is one instead of ten months when the patient is sent to the surgeon. The surgeon also should be prepared to act upon the indications given by the physician. In 1922 a man of 52 was sent to us with a short history of constipation and attacks of colicky pain in the region of the splenic flexure. Nothing abnormal was felt on abdominal examination and an opaque enema revealed no filling defect nor obstruction, but an opaque meal was held up at the splenic flexure for forty-eight hours. The stools constantly contained occult blood. A diagnosis of early carcinoma of the splenic flexure was made, and the patient was advised to undergo operation. Being dissatisfied, he independently consulted a surgeon, who removed his appendix. As no improvement followed, the same surgeon six months later divided adhesions which had formed in connexion with the first operation. In 1925, just over three years after we had diagnosed a growth of the splenic flexure, acute obstructive symptoms developed, and the surgeon, who had ignored our advice, now found an inoperable growth of the colon in the exact position we had originally indicated.

I have said enough to show that the early diagnosis and successful treatment of cancer of the colon depend upon timely co-operation between the general practitioner, the diagnostic team and the surgeon.

Mr. J. P. Lockhart-Mummery

said that if growth in the colon could be treated surgically at an early stage, the results of operation were admirable; he believed them to be better than for carcinoma in any other part of the body. The recurrence rate was low, and the patient was left without disability, and with complete restoration of function. The mortality had been brought down to reasonable limits, considering that many of these patients were physically bad subjects.

The essence of success was early diagnosis. What could be done to improve matters in this respect? Probably only 25 per cent. of cases of carcinoma of the colon reached the surgeon at an operable stage. With regard to the methods of diagnosing suspected carcinoma of the colon, enormous advances had been made during the last twenty years. Twenty years ago it was very rare to find a case of growth in the colon unless the patient had symptoms of acute obstruction. Colotomy was done, and sometimes one could subsequently remove the growth. Now, however, more than half the cases he saw of this condition had not reached the stage of obstruction, and a straightforward anastomosis could be done with very great benefit. This improvement was largely due to the sigmoidoscope, partly to the routine examination, including testing for occult blood, as well as a good interpretation of the skiagrams. X-ray pictures should not be valued as negative, but as confirmatory evidence. When cases had definite symptoms, with or without occult blood, he advised exploratory laparotomy, and as a rule patients agreed if the facts were properly put before them. There was not as yet any serum or other test for the presence of cancer which could be relied upon, though doubtless such a test would be evolved in course of time and would prove of great value.

The onus of getting these cases at a much earlier stage lay largely with the general practitioner, but to a much greater extent with the patients themselves. Often the practitioner did not send cases early enough because he did not examine them properly. But many people had symptoms for which they did not consult anybody, and he did not know how to reach those cases early, especially if they believed that consultation meant X-ray, sigmoidoscopic and other unpleasant procedures. The public must be educated to see their doctor when they had certain symptoms. The general practitioner on the whole realized the importance of the subject, and the number of cases now referred to specialists for expert examination was increasing and would increase; the greatest difficulty was to get the patients to consult their doctor early enough.

Mr. W. B. Gabriel

said that in the United States many insurance companies insisted on a yearly routine examination, and this plan would enable many unsuspected cases of carcinoma to be detected early. During the last few years in his out-patient department at St. Mark's Hospital he had detected at an early stage a number of cases of carcinoma of the rectum, the patients having attended originally with prolapsing piles, or fistula or other minor condition.

Mr. Lionel Norbury

agreed with Mr. Gabriel about the examination of the patient. He might mention two cases of absolutely unsuspected carcinoma of the lower sigmoid. The patients, aged 36 and about 38 respectively, had come to St. Mark's Hospital with a history of recent piles with a little bleeding and protrusion. A routine examination was made of these cases, and in both instances the sigmoidoscope showed early growth in the lower sigmoid which could not be felt.

The situation would not be much improved in regard to early diagnosis until the busy general practitioner could give more help. He believed that practitioners would be only too willing to make thorough routine examinations if time permitted.

Dr. GOULDESBROUGH

said that the oblique view and the double skiagram were not being used as extensively as they ought to be.

With regard to the difficulty, when filling the rectum, of the shadow overlap at the recto-sigmoidal junction, caused by the ballooning of the rectum, he had recently tried using a tilting table to outline the rectum and the sigmoid loop without ballooning. In some cases he had succeeded by keeping the enema pot on a level with the patient's body. By tilting the patient's head down a foot and letting the barium feed trickle slowly into the rectum until the rectum and sigmoid loop are outlined, then by turning the patient in the oblique position one could follow the track of the barium as far as the cæcum without ballooning. When that was impossible he had found it useful to syphon out by lowering the feet of the patient, thus reversing the position, and allowing the material to trickle slowly out of the tube; this procedure was helped by slightly rotating the patient to the right side. The sigmoid loop was then seen clear of the rectum shadow.

Sometimes two or three separate examinations were necessary when observing these radiological appearances of possible obstruction in the bowel, to make certain that they were permanent obstructions. This also gave the operator a good idea of a small stricture, such as that in some of the cases which Dr. Hodgson had shown. Then one could syphon back and syphon in again, to see if the same position was observed.

In differential diagnosis between spasmodic and organic stricture, belladonna was helpful.

The fixation of the colon in any particular position was a very important diagnostic sign of something seriously wrong. It was unusual to find a growth, particularly in the descending colon, unless there was some fixation of the bowel in that position.

Mr. E. T. C. MILLIGAN.

The sigmoidoscopic appearances of adenoma and carcinoma of the colon situated beyond the reach of the examining finger in the rectum are often identical.

For accurate diagnosis the tumour should invariably be palpated before removal in order to test the presence or absence of induration—the distinguishing clinical feature of carcinoma. This may mean opening the abdomen in order to reach the growth, but it should always be done. Insistence on this point may avoid the removal of a rectum for an innocent growth which appeared malignant through a sigmoidoscope.

For routine sigmoidoscopy it is essential that no preparation of the patient be made beforehand, that no aperient be given, and that no bowel wash-out be administered.

Such procedures so change the contents of the bowel that deductions from the appearance of the fæces—quite as valuable as deductions from the naked-eye appearance of the urine in urinary tract disease—are valueless or misleading.

Changes in the fæces produced by highly placed colon growths are quite typical and indicative to the experienced sigmoidoscopist. Even in the absence of X-ray confirmation and other clinical signs of colon growths, the typical appearance of soft, dark, blood-stained mucus-containing fæces, is enough to justify the diagnosis of

growth of the colon and to recommend the serious procedure of exploratory operation. Moreover, with the sigmoidoscope and proctoscope, it is an easy matter to exclude colitis or piles as a reason for the change in the appearance of the fæces, therefore it follows that the unseen growth alone remains as the cause. The reverse also is up to a point true. Normal appearance of fæces seen through a sigmoidoscope is valuable evidence in the difficult problem of excluding the presence of a suspected growth of the colon, undetectable by other methods of investigation.

Thus it is of the greatest importance that the time-honoured custom of altering the appearance of fæces in their natural surroundings by aperients and other preliminary treatment of the bowel before sigmoidoscopy, should be discontinued.

With air inflation it is possible so to distend the bowel that the sigmoidoscope can be directed past and beyond fæces lying in the lumen.

Should one at first be unsuccessful, which is very rare, it is better to examine on another occasion before resorting to aperients and bowel lavage.

Even in patients suffering from colitis and constipation, bowel lavage and aperients, by altering the appearance and position of the bowel contents, rob one of much useful information.

Section of Laryngology.

President—Mr. H. S. Barwell, F.R.C.S.

[*March* 2, 1928.][1]

A Case of Laryngeal Obstruction.

By Philip Green, M.B. (introduced by Mr. C. S. A. Ridout).

E. Y., female, aged 30, formerly a housemaid. History of "laryngitis" from birth, with dyspnœa; lately getting worse; first seen February 2, 1928. Dyspnœa severe on any exertion or on attempted laryngoscopy. Tracheotomy performed February 17. There is only a small triangular aperture patent in the glottis, the vocal cords apparently being covered by a soft-looking membrane, in shape somewhat like an ivy leaf.

Skiagrams and Lantern Slides illustrating Removal of Foreign Bodies.

By H. Bell Tawse, F.R.C.S.

Safety-pin, open, point upwards (which had been in the larynx or trachea for 1½ years) removed from the right bronchus of a child of 2 years 7 months.

I. J., aged 2 years 7 months. When one year old, whilst apparently quite well, had a sudden attack of coughing and choking, accompanied by considerable dyspnœa sufficiently serious to make the mother rush to the doctor; diagnosis, croup.

Hoarseness and cough persisting for twelve months, despite treatment at various places, suggested whooping-cough.

At 2 she developed diphtheria, and dyspnœa necessitated tracheotomy.

First seen by exhibitor at the age of 2 years 4 months, when admitted to hospital with cough and dyspnœa, severe enough to cause cyanosis at times. Position of operation scar seemed to point to a very high tracheotomy, and led to the provisional diagnosis of obstruction from granulations in the larynx.

X-ray photo showed open safety-pin, point up, at level of fifth cervical vertebra, presumably at or just below the cricoid.

Child struggled during administration of anæsthetic; breathing ceased: tracheotomy wound opened up and tube inserted.

In a later skiagram the safety-pin had dropped to the level of the fourth dorsal vertebra, probably at the bifurcation of the trachea.

Under general anæsthesia, pin found in upper part of right bronchus. Point seized, tube pushed down over it, pin and tube removed together.

Pin, originally a gilt one, very corroded, and when laid on a table the pointed branch became separated from the spring.

[1] Discussed at the following Meeting.

64 Bell Tawse: *Skiagrams Illustrating Removal of Foreign Bodies*

Comments.—(*a*) It is not always advisable to attempt closure of an open safety-pin as the initial step towards removal. In this case the pin branch would have separated, leaving two springless foreign bodies—one pointed—to deal with. (*b*) Do chronic hoarseness and spasmodic cough suggest the necessity for X-ray examination in young children?

SAFETY-PIN, OPEN, POINT UPWARDS, IN ŒSOPHAGUS OF GIRL OF 17, REMOVED BY UNINTENTIONAL ENDOGASTRIC VERSION.

The safety-pin had a widespread and a strong spring.

(*a*) The point was disengaged from the œsophageal wall and the tube passed down over it.

Repeated attempts to remove pin and tube together failed, the keeper being invariably caught.

(*b*) The strength of the spring foiled attempts at closure.

(*c*) Version did not succeed.

(*d*) It was decided to pass the pin into the stomach for removal by gastrotomy. As the forceps released the pin in the stomach, it turned over, the spring was seized and pulled into the tube, which was drawn out with the safety pin inside it.

HOSIERY NEEDLE WITH WOOL IN EYE REMOVED FROM LARYNX OF WOMAN OF 25, BY THE INDIRECT METHOD UNDER LOCAL ANÆSTHESIA.

The blunt end was embedded in the right arytænoid, the point in the epiglottis.

My intention was to seize the pin near the blunt end, release it from the arytænoid, push the pointed end through the epiglottis, grasp the point and withdraw the whole needle through that structure. Hosiery needles are very brittle. This one broke in two, and the fragments were removed separately.

HORSE-BEAN BLOCKING RIGHT MAIN BRONCHUS OF CHILD, AGED 7. IN SITU OVER TWENTY-FOUR HOURS. COMPLETE ATELECTASIS RIGHT LUNG.

Patient in considerable distress. Some difficulties experienced.

(1) Swollen œdematous mucosa completely hid the foreign body.

(2) Application of various forceps unsuccessful—all either failed to grasp, or slipped off on tightening.

(3) Suction by cotton-wool and gauze plug—with the idea that sudden withdrawal might create a vacuum, as happens when the piston of a syringe is rapidly pulled up. This failed.

(4) Blunt hook forced past the bean—became inextricably fixed—probably in mouth of small bronchus.

(5) Low tracheotomy—shaft of hook cut through. Hook ultimately disengaged, but did not move the bean.

(6) Forceps again applied through tracheotomy wound, but without result.

(7) Upper end of bean nipped off bit by bit. Rest removed *en masse* by fenestrated forceps.

Comments.—(*a*) The employment of the curved hook created such complications and delay as to make one hesitate to use it again in a similar case. Possibly, if the curve were limited to a right angle it would less likely become anchored, and would be more easily disengaged. (*b*) Removal of the bean by *morcellement* cannot be advocated, but in this case it seemed the lesser of two evils.

I have described this case as "atelectasis," but the radiographer tells me that the skiagram is more like that of an emphysematous lung filled with fluid.

I shall be glad if someone would tell me if this is an instance of obstructive emphysema described by McCrae and Chevalier Jackson. These authorities say there are two types: (1) obstructive emphysema, and (2) obstructive atelectasis, and that these can be differentiated easily by a skiagram. They state that obstructive emphysema occurs in all cases where a vegetable substance is inspired into a bronchus, but that obstructive atelectasis comes on so rapidly when the foreign body is maize or bean, that the one condition passes rapidly into the other, and that in a space of twenty-four hours a "drowned" lung may be found.

Discussion.—Mr. HERBERT TILLEY said that in young children chronic hoarseness and spasmodic cough which did not quickly respond to ordinary measures, suggested the need for radiography. In such cases, should X-rays not give evidence of a foreign body, the bronchoscope should then be used in case there might be an object not opaque to the rays. He had shown before the Section the case of a child who had an open paper-fastener in the left bronchus and it had been there twenty-one months. There had been cough and bronchitic symptoms and the tonsils had been enucleated to relieve the bronchitis, though this was limited to the left lung. When a good radiogram was taken the foreign body was easy to see, and he (the speaker) removed it.

Mr. A. J. WRIGHT said that the heart was displaced towards the affected lung, suggesting that the lung was in a state of collapse, and not of emphysema.

Mr. W. M. MOLLISON said that, illustrating the difficulty sometimes experienced, he recently had in hospital an infant, aged 1 year, who had been under a physician for about a month, and had received active treatment for bronchitis for a fortnight, but with no good result. As so much laryngeal noise was made by the patient and disturbed the rest of others in the ward, he (the speaker) was asked for an opinion. He found nothing wrong with the larynx, and suggested a skiagram. This showed a collar-stud just below the cricoid; it had been there six months. An ordinary small tube was passed into the œsophagus, but it was impossible to remove the collar-stud, therefore he had a small Mosher's tube made; even then it was difficult to remove the stud, as it had button-holed itself between the œsophagus and the trachea. However, he managed to do so, but opened the fistula between the trachea and the œsophagus, and the child died.

Mr. H. S. BARWELL (President) said he had had a case similar to Mr. Mollison's, but it occurred long ago when the direct method was first introduced. A metal collar-stud was fixed like an anchor in the upper aperture of the œsophagus under the prominence of the cricoid cartilage; he had failed to remove it and the infant had died. As it had been *in extremis* when brought to hospital, no external operation could be attempted.

Sir JAMES DUNDAS-GRANT said that a hook had been made, after the fashion of Lister's ear hook, and like certain bottle-cork extractors which were passed on the flat beyond the cork and then rotated through a quarter turn so as to catch it. He had found this hook very useful in "orientating" and loosening the large portion of an inferior denture [shown on the screen] which he had removed by means of D. R. Paterson's forceps furnished with Irwin Moore's blades. It was at the level of the clavicle.

Mr. BELL TAWSE (in reply) said the hook he used was the Chevalier Jackson hook. He believed there was now on the market a new one, designed by Birkett, with the hook at a right angle, and with a mark on the handle so that one could tell what was the exact position of the hook at any time.

Mr. H. S. BARWELL (President) said that Chevalier Jackson had come to the conclusion that no hook should be at as sharp an angle as a right angle.

66 Dundas-Grant: *Excision of Ventricular Band*

Case of Tuberculosis of the Ventricle of the Larynx, treated by Partial Excision of the Ventricular Band.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

A MAN of middle age, with tuberculosis and extreme hoarseness, was found to have a rounded, fleshy outgrowth protruding from the anterior two-thirds of the right ventricle. This was punctured with the galvano-cautery on several occasions with some benefit. In August, 1927, I removed with punch-forceps a portion of the middle of the ventricular band, since which the hoarseness has diminished with great rapidity, the patient voluntarily stating that he can now sing.

I have made a notch in the ventricular band in several other cases in which there seemed to be a focus of tuberculous disease in the ventricle.

Mr. H. E. BARWELL (President) said that these cases of tuberculosis inside the ventricle presented special difficulties for local treatment. In one case he had performed laryngo-fissure and removed the whole of the ventricular band, and the patient's general condition had improved considerably.

[*May* 4, 1928.]

Some Varieties of Skin Flaps in Connexion with Cases of Total and Hemi-Laryngectomies.

By R. GRAHAM BROWN, F.C.S.A. (Brisbane).

THE following cases seem worth recording on account of the successful attempts to improve the surgical technique in the regions of the larynx and trachea.

I.—*Quadrilateral Skin Flap in Total Laryngectomy.*—The advantage of a quadrilateral skin and platysma flap, as recommended by Professor Tapia of Madrid and others, in the operation of extirpation of the larynx, may be questioned over the ordinary "T"-shaped one. It is, however, our duty in these cases to prefer a procedure likely to give a primary wound closure than to run an extra risk of non-closure for the sake of the questionably less disfiguring scar of the latter incision. Most of us have seen troublesome pharyngostomes following laryngectomies. It is assumed that all care has been taken in the approximation of the pharyngeal mucous membrane and the muscle layers. The inclusion of the platysma fibres in the skin flap is, I think, an obvious advantage. I have adopted the rule to approximate the mucous membrane by a purse-string suture, wherever possible, which brings the submucous tissue in apposition to bring together the muscles in layers by single catgut sutures and to use no drainage. In my last three cases, where this procedure was followed, the patients were fed with a catheter through the mouth after forty-eight hours and were able to swallow fluid foods three days later.

II.—*Preliminary Tracheotomy in Total Laryngectomy.*—During the period of after-treatment of a case in which a low preliminary tracheotomy has been performed I have been, like others, troubled with the unclean state of the wound and with the formation of granulations and subsequent scar, the results of an almost futile endeavour to bring the trachea forwards towards the skin. Especially is this the case when one follows the procedure of separating the trachea and œsophagus by means of gauze packing (I now use acriflavin strips for this purpose instead of bismuth and iodoform gauze). I considered that it would be easier and of more advantage to slide the skin deep down into the neck wound and fix it there. This can be done very readily by undermining the skin over the sterno-mastoid muscles, sliding the flaps inwards and by means of a mattress suture on each side, which passes in turn through the skin near its cut edge, the ribbon muscles, the sterno-mastoid muscle and

comes out through the skin again, some 1½ in. to 2 in. external to the cut edge, fix it there. The skin rests in position without any dragging, and the cut edge is separated from the trachea only by the thin layer of gauze packing. No sutures are used between the trachea and skin and the wound is "clean." Following the procedure laid down by Sir StClair Thomson and Mr. Wilfred Trotter, I excise a large disc from the anterior wall of the trachea when performing a tracheotomy in these cases. The possibility of stenosis of the tracheo-cutaneous wound must always be kept in mind; and by using a skin "slide," as suggested, this possibility will be better provided against. In this case the large size of the funnel opening to the trachea might be criticized, but this, as one anticipated, narrowed somewhat. The opening was purposely made over-large, because of the fact that the patient lives in the country, some 300 miles distant by train, and is not in a position to be inspected by me at short intervals. In any case it is, if a fault, at all events one in the right direction (fig. 1).

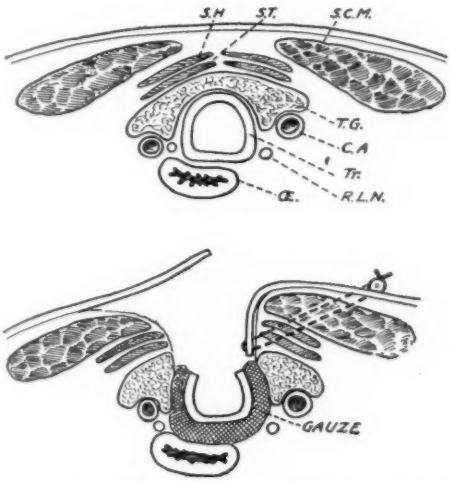

FIG. 1. *Case II.*—Diagrammatic representation of operative procedure showing large opening and skin flap, dissected up on left side and in position on right side.

III.—*Reconstruction of Larynx.*—The next case illustrates stages in an attempted reconstruction of a larynx in a girl aged 16. History: when she was aged 2½ years the patient had undergone an emergency tracheotomy for laryngeal diphtheria by a general practitioner in the country. Her parents stated definitely that since this illness she had not been able to talk, or to breathe through the larynx. Six months before she was brought to me an attempt had been made to excise the scar tissue. There was a large laryngeal fissure; there was also total stenosis at the level of the vocal cords, and there was much intra-laryngeal and external scarring (fig. 2). The vocal cords and arytenoid cartilages were involved in the scarring, but there was some movement of the right arytenoid and the right vocal cord. No movement could be seen on the left side. The girl had œsophageal speech, but this was weak, and was associated with "clicks" and "clacks" which masked it. She had really never learned to talk through the larynx, and one kept this in mind in noting the amount of movement of the laryngeal parts, the appearance of any movement at all promising a favourable development in this direction. In order to deal effectively with the scar tissue it was necessary to perform a hemi-laryngectomy. There was such a degree of calcification of the cricoid and thyroid cartilages, and they were so fused together that it was necessary to

68 Graham Brown: *Skin Flaps in Total and Hemi-Laryngectomies*

remove the left half of each of these structures together with part of the left arytenoid cartilage. The calcification was so hard that bone forceps had to be used to remove it. A transverse skin flap was dissected up from the supra-hyoid region, and the free end turned down into the larynx where it was fixed, without tension, by means of three fine catgut sutures. Two months later the anterior wall of the larynx was re-formed by cutting across the base of the flap and turning it down. This, in turn, was covered by "slide" skin flaps made by undermining the skin on both sides and suturing in the middle line. The pharyngeal end of the cast was purposely made long and pointed to allow of its smooth introduction, in order to avoid, as far as possible, damage to the skin graft surface. After the hemi-laryngectomy operation there was some slight escape of saliva, but this soon disappeared. The patient is now able to breathe through the larynx during the whole of the night, and her speech is a medium loud whisper. Considering the fact that she has, to all intents and purposes, never learnt to phonate, and does not know the value of the accessory resonators, it will necessarily

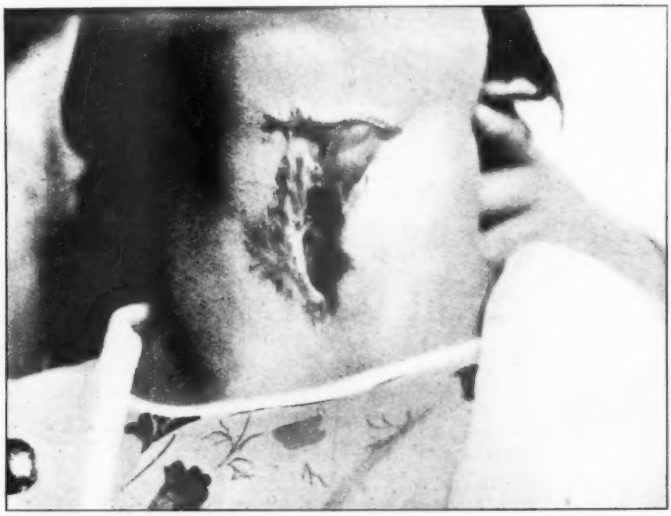

FIG. 2. *Case III.*—Showing large fissure, the result of an attempt at excision of a stenosis of the larynx.

be some time before she acquires a strong voice. One can reasonably hope for a fair voice after some contraction has taken place, especially if the right vocal muscles develop, with exercises, after she has learnt to coördinate the expiratory efforts which are essential for speech. I believe, too, that the airway will be maintained to a degree sufficient to allow of hope that eventually the tracheotomy tube may be discarded. Four and a half months elapsed between the first and last operations.

In conclusion, it may not be out of place to state that the operative procedures in these three cases were all performed under local analgesia, or rather a form of synergetic analgesia, a method which I consider ideal for this class of operations.

Mr. HERBERT TILLEY said that all teachers should impress upon their students the necessity of always performing a low tracheotomy. Most of the trouble in the case which had just been narrated was due to the surgeon having practically performed laryngo-fissure.

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Bronchiectasis in Children: The "Pseudo-robust" Appearance in Cases associated with Nasal Accessory Sinus Suppuration.

By R. GRAHAM BROWN, F.C.S.A. (Brisbane).

I SHOULD like to draw attention to the frequency of the association of antral (and more rarely other nasal accessory sinus) disease with bronchiectasis in children. In Queensland there is a considerable amount of nasal accessory sinus infection, and, in consequence, those working there are constantly on the look-out for this affection. This condition is possibly more prevalent in Great Britain than one would gather from statements in text-books and elsewhere. However, in this matter we, in Queensland, where there is a dry and dusty atmosphere, may possibly have a greater experience than practitioners in Great Britain, where the reverse climatic conditions hold.

For convenience I classify cases of bronchiectasis in children into two groups:—

(1) Those associated with and probably dependent upon antral—or, more rarely, other accessory sinus—suppuration.

(2) Those not so associated, subdivided into: (*a*) Those associated with a foreign body (opaque or non-opaque to X-rays) in the bronchial tree. (*b*) Those due to other causes, such as, following pneumonia, influenza, etc.

With the latter sub-group, 2 (*b*), we have here little concern except that we may be called upon to inject "iodized oil" into the bronchi to define the extent of the cavity formation or to help in the diagnosis of a lung disease, etc. In 1921, I drew attention to this question,[1] pointing out that there was a typical facies in these cases of bronchiectasis with antral suppuration in children. So much is this so, that, when such a facies in children is associated with a loose, rattling cough, it is suggestive of antral suppuration with bronchiectasis. Several patients were shown at the meeting at which the paper was read to illustrate the point. These little patients appear on casual observation very healthy, whereas they are really far from well and I prefer to use the term "pseudo-robust" with regard to them. From about the age of 9 or 10 onwards this pseudo-robust appearance tends to be replaced by a drawn and sickly countenance and the patient then looks chronically ill. The parents frequently say that they cannot understand why the child looks so well and yet his health is really "up and down," more "down" than "up." The child has a cough, usually slight, but it is chronic and frequently disturbs him at nights. He is subject to feverish attacks and to recurrent "colds," bronchitis, etc. He appears to be fat, but in reality he is flabby. The history generally is that the cough developed soon after an attack of whooping-cough or some such ailment, occurring in the first eighteen months or so of life, and that it has been present with exacerbation ever since. Seldom is any mention made of nasal trouble except possibly that the child blows his nose frequently and is subject to colds. Several children have been brought to me suffering from chronic suppurative otitis media, when a loose cough has been noticed and upon further investigation bronchiectasis with sinus suppuration has been discovered. Upon hearing a loose cough, however slight, in a "pseudo-robust" child, I am immediately suspicious of bronchiectasis and proceed to exclude it. My practice upon hearing such a cough is to make the child cough strongly (a type of cough which he is not in the habit of performing) and bring up, if possible, the secretion. It is surprising how difficult it is to get even young adults to cough sufficiently hard enough to bring the sputum as far as the mouth and it is sometimes necessary almost

1 *Med. Journ. of Australia*, 1921, i, 445 ("B.M.A. News").

to "bully" the patient into doing it. The reason for this is, I think, that the patient has been used to coughing just sufficiently hard to get the sputum out of the larynx. whereupon he immediately swallows it. A good plan with these children is to hold them upside down and then to make them cough. Upon the appearance of a thick sputum most parents have expressed great surprise and have assured me that they have never seen any similar sputum from the child before. All these children, excepting those with the typical adenoid facies, which is a rare accompaniment, show well developed, broad faces. In many instances this appearance could be well styled "over-development of the width of the face." It is, of course, due to the large size of the antral cavities and, consequently, of the upper jaws. I hold the view, at present, that pus in the antra of young children expands the upper jaws to a greater degree than does air in its normal passage through the nostrils and air sinuses. Contrast this condition with that found in the "adenoid child" in which case sufficient air has not passed through the nostrils and sinuses. I have only on rare occasions seen chronic bronchiectasis and sinus suppuration together, in the adenoid type of child and then only in older children. I consider that the affection has developed after the first eight to ten years or so—i.e., after the presence of adenoids has well established the improper development of the upper jaws. Practically, whenever there was any suspicion of sinus disease I have found pus (although it may be only slight in amount) in the nose and there is a varying amount of nasal obstruction, particularly in the middle turbinate regions. Adenoids and septic tonsils too are invariably present. The amount of pus to be seen is frequently small and if the patient has blown his nose just before being examined (a usual happening) very little or possibly nothing of this nature may be seen. As a matter of routine all these cases are sent for X-ray photographs of the sinuses and the chest, I insist upon stereoscopic views of the sinuses, as an exposure, in one plane only, has practically no value. Much can be learned from a good stereoscopic picture. A picture of the chest does no more than tell us that there is a suspicion (be it great or slight) of bronchiectasis, but the chest screening gives details of movement, etc. We, however, get no details as to sizes of cavities and such-like information and I am therefore strongly of opinion that the consultant should be present at such screenings.

As it is essential in the proper treatment of these cases to correct any nasal obstruction, particular attention must be paid to this factor. Much can be done for these children by clearing up the antral and any other possible accessory sinus suppuration. In dealing with the antra, conservative measures should first be tried. However, should anything in the nature of adenoids or a nasal obstruction—particularly in the middle turbinate regions—be present, these should be rectified in a radical manner. In those cases in which there is no middle turbinate obstruction—a rarity, I have found in this condition—but a mere swollen mucous membrane producing obstruction to the ventilation of the sinuses, the simple removal of the adenoids may be followed by a clearing up of the antral suppuration. The question is often asked how old a patient should be before he is subjected to surgical interference with the nasal septum, middle turbinates, etc. I do not think that this requires an answer. The important question is how to eradicate the bronchiectasis, which is due, most probably, to a simultaneous—or more likely a pre-existing—and still present sinus suppuration. As the majority of cases of chronic antral suppuration in children can be cleaned by "washings-out," repeated at intervals, and as this is dependent upon proper ventilation of the sinuses, the only logical course to follow is to do whatever is necessary to get rid of the middle turbinate outward pressure, which is invariably present. It may be necessary simply to do a

middle turbinectomy (with tonsillectomy and adenoidectomy if required at the same sitting), or it may be necessary to correct, in a conservative manner, an early septal deflection. I have no hesitation in these cases in operating upon the nasal septum, for it is better to run the risk of a possible—though not probable—slight nasal deformity in later years than to leave a condition which, if untreated, will eventually become a pitiable state.

It is futile to treat the bronchiectasis without checking the sinus suppuration. The antra are washed out at the time of the removal of the tonsils and adenoids and/or the middle turbinate or septal obstruction. This, in young children, I have made a practice of doing through the anterior wall. Repeated antral "washings" after providing good ventilation in the middle meatus generally give good results. One may get the antra clean, but there may be relapses following subsequent colds. The "washing-out" can then be repeated. Repeated colds, however, after correction of a middle turbinate obstruction, are not the rule, although such a correction does not by any means give freedom from colds. Even in young children it is possible to wash out the antra comfortably under local analgesia, although I generally prefer a general anæsthetic for the first occasion, and this can be done at the time of the rectification of the nasal obstruction. Subsequent "washings-out" are then performed, if possible, with local analgesia. The method of approach to the antrum which I employ in young children is through its anterior wall, and this, when using local analgesia, follows an injection of about 0·5—1 c.c. of 1 per cent. novocain solution at the point of exit of the infra-orbital nerve; the injection and subsequent puncture being made under the lip. To puncture the antra of a young child through the nose is, in most cases, an unsatisfactory proceeding and in some cases even with the aid of a general anæsthetic almost impossible without decided risks. At subsequent attempts it is easy to find the original perforation in the anterior wall of the antrum. Should the antral suppuration not clear up, an intra-nasal operation upon the sinus is indicated, and if this fails a radical operation should be performed. These operations, however, I have found rarely necessary. Certainly there is no need to hurry the radical operation. Recently it was my unpleasant task to perform a radical antral operation upon a bronchiectatic child under four years old, in whom, fourteen days after obtaining a clear return with antral lavage, an acute exacerbation with phlegmon of the cheek developed. Although so young she had a surprisingly large antrum which resembled an adult one in development.

In the second group, consisting of cases due to a foreign body in the bronchial tree, there is a favourable prognosis after removal of the foreign body, but I must confess that the finding of a non-opaque foreign body may be at times very difficult.

Regarding treatment of the bronchiectasis, the procedure is mainly upon general lines and the practitioner should avoid the use of respiratory sedatives; he should, indeed, use stimulating drugs. The best method I have found of treating these cases is by means of posture and the creosote vapour baths. Drainage by posture accompanied by frequent coughing is an excellent method of emptying the cavities, and it should be used as a routine. The positions to be assumed can be worked out after the details of the cavities and the positions of their outlets, etc., have been demonstrated by iodized oil bronchial injection[1] (I have recently employed a French preparation named "oliolase," less viscid, and hence more easily expressed from the syringe than lipiodol, and equally efficient for giving a shadow). Drainage by posture should be performed regularly and frequently during the day. The use of the bronchoscope, as a means of drainage and lavage of the

[1] *Med. Journ. of Australia*, 1927, ii, 506.

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bronchiectatic cavities, does not produce the benefits promised a few years ago, and at the Hajek Clinic (Vienna) seems to be going out of favour. The X-ray photographic results of injection of iodized oil in young children are not satisfactory, as the injection must be done under general anæsthesia, and it is almost impossible to get good pictures under these conditions owing to the uncontrolled respiratory movements and to the rapid heart beats. It is useless, moreover, to allow the patient to regain consciousness from an anæsthetic and then take the pictures, for the vomiting, usually associated with the anæsthetic, by ejecting the oil, nullifies one's efforts. However, if the surgeon is present at the "screening" he can learn a great deal concerning the position, form and extent of the dilatations. In children of 8 or 9 years of age and upwards, injections of iodized oil through the glottis may, with the aid of kindly encouragement, generally be done under local analgesia. The group which undoubtedly shows the best results is that in which a foreign body is present in the bronchi. In these cases the manner in which the symptoms and physical signs may clear up is remarkable.

These cases of bronchiectasis in children are coming more frequently under our notice, and as the state of these little ones, if the condition is allowed to drift, becomes pathetic, more care should be taken to eliminate the presence of bronchiectasis, acute or chronic, in all children suffering from chronic coughs, and early treatment of the cause should be energetically undertaken. Although an absolute cure can really never be promised, the chest condition, after efficient treatment of the cause has been carried out, may improve so greatly as almost to warrant it being called a cure. However, this much is certain, we can, at least, bring about by early and vigorous treatment, an amelioration, sometimes very considerable, of the lot of these unfortunate children.

Discussion.—Sir JAMES DUNDAS-GRANT said that this communication had forcibly impressed on members the facial characteristics of children suffering from bronchiectasis. He (the speaker) had previously had some idea of them from seeing children with the "pseudo-robust" condition at Brompton Hospital, and, in consequence, guessing that those children had bronchiectasis.

Mr. M. VLASTO asked how Mr. Graham Brown explained this spurious robust appearance. These cases seemed to be exactly like other cases of chronic toxæmia. He had had no personal experience—apart from the inspiration of foreign bodies—of bronchiectasis originating from infection of the accessory sinuses of the nose. It was peculiar that amongst the few cases of infected antra in children there should occur here and there a case of bronchiectasis, and yet amongst the thousands of children with highly infected adenoids bronchiectasis should be almost unknown. His daily experience was that of a child brought up with a chronic cough, the diagnosis of infected tonsils and adenoids was made, the operation was performed, and sooner or later the cough cleared up. Possibly in some of these cases the antrum on proof puncture might be found to contain pus.

Mr. J. B. HORGAN said he thought the point which Mr. Graham Brown wished to emphasize was that he systematically sought for trouble in the nasal sinuses in children, whereas perhaps most members did not. Those who did would probably agree with Mr. Graham Brown who was to be congratulated on the advance he had made in the surgery of nasal sinusitis in children, a subject about which there was yet a good deal to be learnt.

Dr. J. S. FRASER said that with regard to the removal of the middle turbinal, he wondered whether Mr. Graham Brown had tried to clear out the ethmoid and so ventilate the maxillary antrum without removing the middle turbinal. In Halle's operation, which he had found successful in certain cases, the middle turbinate was fractured towards the septum, and the ethmoid was exenterated. If the middle turbinal could be left it was an advantage.

With regard to the septal operation in children, he recalled the case of a boy who had frequent attacks of bronchitis and asthma; the septum blocked one side of the nose partially, the other side totally. Though the operation cured both ailments, the boy's mother was still resentful with regard to the shape of her son's nose.

Mr. E. D. D. DAVIS said the method of puncturing the canine fossa was admirable; he wished to know what kind of trochar Mr. Graham Brown used, and whether he had had any adverse results. The author seemed to consider that nasal sinus suppuration was a cause of bronchiectasis; this would be difficult to prove.

Mr. ANDREW WYLIE agreed that the antrum should be thoroughly investigated, especially in children always complaining of nasal catarrh, or those suffering from decayed teeth. When Dr. Watson-Williams had brought this fact to the notice of the Section several years ago, several Members, including himself (Mr. Wylie), had considered the condition somewhat exaggerated. He had since changed his mind, having in several cases found pus and caseous matter in the antrum.

Mr. GRAHAM BROWN, in reply to Mr. Vlasto, said that the children were not robust, but *pseudo*-robust; they were really flabby, and did not run about and play with zest in the manner of healthy children. All his patients were not Australians; one boy, who had advanced bronchiectasis, came from London. In answer to Dr. Fraser, he had no hesitation about performing the operation of middle turbinectomy, but he was strongly against interfering with the inferior turbinate. He performed a middle turbinotomy to give ventilation to all the air sinuses which opened under the middle turbinate, and he only removed just sufficient of that bone to give the required ventilation. It might be necessary to do a submucous resection in order to deal efficiently with the middle turbinate. He left a fair margin of the septal cartilage anteriorly. There were times when the operator must be guided by his conscience, and perform a septal operation when necessary. In reply to Mr. Davis, to perforate the anterior wall of the antrum no special type of needle was needed; he used one of 1·5 to 2 mm. diameter. It was a simple matter to pass the instrument through the bone when one kept immediately beneath the infra-orbital foramen, but care was needed, because if one went laterally one met with thick bone. Regarding the child in whose case he had been obliged to perform a radical operation, after several washings out, he had concluded that the antra were clean. However, fourteen days after the last wash-out the patient had a marked "flare up" and became seriously sick. He was forced to open the antra. The resistance of these bronchiectatic children was lower than that of normal children. He could not say definitely that the sinus suppuration was the cause of the bronchiectasis, but it existed at the same time, and in these cases there had almost always been evidence that it had existed previous to the bronchiectasis. He believed that in these cases the bronchiectasis was secondary to the antral suppuration, and he was definitely of opinion that the antral suppuration prevented improvement with regard to the bronchiectasis.

Fixation of Left Vocal Cord as the Result of Acute Rheumatism.

By W. M. MOLLISON, M.Ch.

R. S., AGED 16, had an attack of chorea and acute rheumatism six months ago; during the illness partial loss of voice was noticed and has persisted.

The left cord appears fixed.

Discussion.—Mr. HERBERT TILLEY said this was similar to a case which he had described at the Edinburgh Meeting of the B.M.A. last summer, that of a doctor with an inactive left cord and œdema of the corresponding arytenoid, who had had an attack of acute rheumatic fever. He (Mr. Tilley) had seen the patient two months later and found that the action of the cord was normal and the œdema had quite disappeared.

Mr. E. D. D. DAVIS wondered whether Mr. Mollison had excluded recurrent laryngeal paralysis. If not, that might be the cause of the fixation.

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Chronic Right Frontal Sinus Empyema with very Severe Headaches cured by External Operation after Failure by Intranasal Method of Drainage.

By HERBERT TILLEY, F.R.C.S.

MR. A. V. M., aged 29, had had several attacks of severe and incapacitating right-sided frontal headaches for the past ten years, associated with "nasal catarrh." The attacks lasted for about four days, and the pain was intense for about one hour each morning. Patient said he had suffered from "angio-neurotic œdema of testicles, foot, palm of hand and uvula."

Examination.—A prominent septal spine was pressing against the anterior region of the right middle turbinal. Muco-pus was seen in anterior cleft of middle meatus, and beyond this the mucosa was polypoid. A considerable mass of adenoids in naso-pharynx. Maxillary sinuses clear on transillumination.

August 4, 1927.—Under general anæsthesia right middle turbinal removed and also the "lateral mass" of the ethmoid, which was in a state of polypoid degeneration. Anterior wall of right sphenoidal sinus removed and the fronto-nasal canal enlarged with author's rasps. Adenoids removed.

The symptoms were only partially relieved, so the patient was readmitted on November 23, 1927, and the external operation was performed. The frontal sinus was lined with œdematous mucous membrane and it contained pus. A free opening was made into the nasal cavity and a drainage tube inserted, leading from the upper end of the enlarged fronto-nasal canal to the external naris. The edges of the external wound were sutured. The tube was removed on the fourth day.

The patient made a rapid recovery, and has had no recurrence of the headaches or of the discharge. The scar is scarcely noticeable.

Bilateral, Chronic Suppuration of the Frontal Sinuses with Subperiosteal Abscess over the Right Sinus. Cured by External Operation.

By HERBERT TILLEY, F.R.C.S.

MRS. F., aged 48. Admitted to the Royal Ear Hospital (University College Hospital) for "swelling over the forehead for one month, pain (frontal) for ten weeks, and sleeplessness due to pain."

Examination, March 26, 1927.—History of tuberculosis of both lungs which has been arrested. A tense, painful and fluctuating swelling about the size of half-a-crown in the lower central frontal region. Tissues immediately round the swelling slightly œdematous.

Transillumination right antrum dull. Purulent discharge could be washed from right frontal sinus, and during the irrigation the external swelling increased in size, and conversely, by means of a suction bag fixed to the cannula the swelling could be made to diminish in size.

Treatment.—For three weeks the sinus was irrigated daily with mild antiseptic lotions and 15 per cent. solution of argyrol was injected, but only moderate relief of symptoms was obtained.

On June 21, 1927, external operation on the right frontal sinus revealed a fistula in the anterior wall of the sinus, and the surrounding bone was inflamed and easily

removed with forceps. The septum between the right and left sinuses was absent, and both were filled with swollen pyogenic membrane. Both sinuses were freely exposed, a portion of the anterior walls was removed, the fronto-nasal canals were enlarged and iodoform gauze drains were inserted into the sinuses. The outer thirds only of the skin incisions were sutured.

Daily dressings were carried out until the sinuses were filled with healthy granulations and the incisions were allowed to close.

The patient was discharged cured on July 22, 1927.

The type of case is rather unusual in that: (1) The perforation of the anterior wall of the right frontal sinus seems to have developed slowly; (2) the patient is a tuberculous subject, and still has physical signs indicative of old bilateral pulmonary tuberculosis which appears to have been arrested.

Discussion.—Dr. J. S. FRASER said that if one made the incision right through the middle of the eyebrow and took a bolder curve down the side of the nose, the scar was even less noticeable than if the incision was behind and below the eyebrow.

Mr. NORMAN PATTERSON said he did not agree with Dr. Fraser. If the incision was made through the eyebrow and a depressed scar resulted, the deformity was greater than if it were made below the eyebrow.

Mr. STIRK ADAMS said he himself had undergone an external frontal sinus operation five years ago, the incision having been made through the centre of the eyebrow, and he thought Members would have difficulty in deciding on which side the scar was.

Mr. TILLEY (in reply) said that formerly many external operations were done on the frontal sinus but they had been to some extent supplanted by the intranasal route. When, formerly, he operated externally, he used to make the incision through the eyebrow. It was true the scar was not visible, but the eyebrow tended to become narrowed on the side of the scar and in certain complexions this was noticeable. For that reason he now incised immediately below, so that the eyebrow was left the same width as that on the other side.

Post-mortem Specimen of Portion of Œsophageal Wall from Case shown November 4, 1927. ("Achalasia of Cardia after treatment by Hurst's Mercury Tubes.")

By HERBERT TILLEY, F.R.C.S.

THE patient was re-admitted to University College Hospital, February 27, 1928, with a history of broncho-pneumonia for four weeks, and after the acute symptoms had subsided he began to expectorate large quantities of offensive sputum.

On admission the patient was pale, wasted, and apathetic; only by a strong effort could he whisper, and even this caused increase in his breathlessness. Temperature 99° F. Pulse 88. Physical signs pointed to consolidation of left base. No difficulty in swallowing. Patient died on March 2.

Post-mortem.—The left lower lobe of the lung contained an abscess cavity filled foul pus and necrotic lung tissue. No foreign body. In the lower lobe of the right lung with were several small abscesses and one large one filled with similar material to that in the left lung abscess.

There was no sign of obstruction in the œsophagus, and its wall was intact, but the mucous membrane showed a patchy hyperplasia.

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I am indebted to Mr. Barnard (Curator) for the following report:—

"The œsophagus is greatly dilated, measuring up to 9·5 cm. in circumference, and the muscularis thickened (up to 0·4 cm. thick). The mucosa is profusely covered with scale-like pearly-white, thickenings which, on microscopic section, prove to be patches of epithelial hyperplasia. Except in the region of the thickened patches, the mucosa is everywhere the seat of a chronic inflammation."

Dr. J. S. FRASER said he thought it might be worth while to examine the cells of Auerbach's plexus in this case. Dr. Cameron, of Glasgow, recently demonstrated beautiful preparations from cases of dilatation of the œsophagus, showing small-celled infiltration round the cells of Auerbach's plexus and degeneration of the ganglion cells. It was recognized even by Mosher, of Boston, that the probable pathology of achalasia or dilatation of the œsophagus was this affection of Auerbach's plexus. Previously Mosher held entirely different views regarding the pathology of the condition.

Section of Comparative Medicine.

President—Sir D'Arcy Power, K.B.E., F.R.C.S.

[*March* 28, 1928.]

Intradermic Tuberculin Testing in Cattle.

By T. Dalling, J. H. Mason, and W. S. Gordon.

We have carried out many tests in cattle, using concentrated tuberculin intradermically, and some of the points investigated have already been published.

Two herds of milch cows have been under our observation for the past three years, and, using the double intradermic tuberculin test twice a year, we have succeeded in separating reactors from non-reactors and in keeping the non-reactor sections tubercle free. Farm A had 103 milk cows and heifers, and, when tested in 1925, sixty-seven reacted positively and thirty-six gave a negative result. From the non-reacting cows, six were chosen for the supplying of milk for calf-rearing and removed to a special calf-rearing farm; thirty-one of the best cows were removed to a new farm. These cattle have been tested twice annually for the last two and a half years and those remaining in the herds are still non-reactors. It has been necessary to add to these herds: double intradermic testing has always been carried out on these additions before their admission to the herds.

Farm B had ninety-six milk cows and heifers. Three years ago, application of the double intradermic test showed that fifty-eight were affected with tuberculosis.

The thirty-eight non-reactors were dealt with as on Farm A, except that for one and a half years they were kept on the same farm as the reactors but in separate sheds. An occasional reactor was detected in subsequent tests, but one and a half years ago the owner took over a new farm to which the non-reacting cattle were sent: since that time no further reactors have been noted. Additions to the herd are dealt with as on Farm A. Subcutaneous tests have also been done on two occasions and the results agree with the double intradermic tests.

Calf-rearing on these two farms has been under our observation for three years. The farmer, unfortunately, insists that the calves suck their dams once; they are then removed to the farms on which the non-reacting cattle are kept and are fed on milk from these cows. Intradermic tuberculin tests have been carried out on these calves at regular intervals, and of over 100 calves so reared only two have shown reactions, while the majority of control calves kept on the original farms and fed on milk from the reacting cows have reacted at some stage in their lives to the tuberculin tests. It is then evident that the double intradermic test is a satisfactory test to employ in the cleaning up of a herd and in the testing of cows supplying milk for the feeding of calves on a presumably clean farm.

In addition to the cleaning up of herds, we have studied the reactions produced by the double intradermic method of testing on other cattle, and it may be of interest to note the following:—

(*a*) *Standardization of Tuberculin.*—The tuberculin used for the tests has been standardized by the intradermic guinea-pig method, and we are convinced that a tuberculin so standardized and passed as equal to the Frankfurt standard, is an efficient preparation for intradermic use in cattle.

Tuberculous cattle will not show the same degree or type of reaction in every case with the same tuberculin, in fact similar amounts of the same tuberculin, injected into different parts of the skin of the same animal, produce different

reactions, but experience in reading reactions will cause one to classify different degrees and types of reaction as positive.

(*b*) *Strength of Tuberculin.*—While most of our tests have been made with tuberculin equal in potency to the Frankfurt standard, lately we have used tuberculin of double and even quadruple strength. In our experience, a cleaner cut reaction is obtained by such a preparation. We have had the feeling that a strong tuberculin, while giving clearer positive reactions, produced some degree of non-specific reaction even in non-reacting cattle, and that the more marked reaction in tuberculous cattle was due to this non-specific reaction. Our numerous tests, however, convince us that, allowing for an increase in non-specific reaction, a strong tuberculin gives a more definite and more easily read positive reaction than one of standard potency. In all our experience there have been only four cattle in which a very marked degree of difference in reaction has occurred when a standard and a fourfold strong tuberculin were used, and it is very probable that, using tuberculin equal to standard, these cows would have been passed as non-reactors. We believe that the benefit derived from the use of this strong tuberculin is that there will be fewer indefinite reactions recorded. Indefinite reactions do occur and some cattle will continue to show such indefinite reactions at all tests, no matter how often applied. With the use of strong tuberculin these indefinite reactions are reduced in number but are still observed. It may be that when purified tuberculin has been thoroughly worked out and is available for general work, the "indefinite reaction" will be further reduced or will disappear entirely.

(*c*) *Comparison of Subcutaneous and Intradermic Tests.*—We have tested many cows and heifers by both methods, usually leaving an interval of about one month between the two tests, and as a rule the results are in very close agreement. We have, however, records of the testing of cattle in which exactly opposite results have been recorded. In one cow only have we observed that positive reactions were obtained by the use of tuberculin subcutaneously, while the intradermic tests were negative. This cow was tested on many occasions and never once did she react to the intradermic tests, while on every occasion a typical temperature chart followed the use of diluted tuberculin subcutaneously. This cow died, and post-mortem examination showed advanced generalized tuberculosis, which was confirmed by guinea-pig inoculation.

In five cows only are we able to record the reverse, viz., they reacted to the intradermic test while subcutaneous tuberculin gave no reaction. These cows were tested intradermically and a month later received a dose of tuberculin subcutaneously. They reacted typically to the first test but gave an entirely negative reaction to the second test. In six months' time they were again tested by both methods, one month elapsing between the tests, and the results were as before. These cows are now isolated and will be subjected to further testing in the near future. It should be noted that a double dose of tuberculin was used for the subcutaneous test and temperatures were taken every three hours after injection.

Cases of similar results are recorded in the literature, but after a series of over 300 tests by both methods these five cows are the only ones which in our hands have yielded such results.

The conclusion one would draw from the large series of tests we have carried out is that the double intradermic method of tuberculin testing is reliable and the amount of error is probably somewhat smaller than when the subcutaneous method is used.

Discussion.—Dr. LESLIE SHEATHER asked how the quadruple strength concentrated tuberculin was prepared.

Dr. F. C. MINETT asked whether, in Mr. Dalling's experience with the double intradermal test, it had not been observed that young tuberculous bovines reacted more intensely than older ones.

Major-General SIR JOHN MOORE asked Mr. Dalling whether, in view of the fact that the standardized tuberculin gave no reaction and the tuberculin of quadruple strength did, he considered the former tuberculin was perfectly standardized or not.

Mr. GEORGE W. DUNKIN: Mr. Dalling has stated that when cows which had reacted to the double intradermal tuberculin test give birth to calves these calves were permitted to suck their dams once only and were then removed to tubercle-free premises. Was it to be assumed therefore that the colostrum of tubercular cows did not contain the tubercle bacillus?

Major DALLING (in reply) said that in his experience the degree of reaction among young tuberculous bovines was neither greater nor less than that observed among older animals. The standard tuberculins used for comparative tests had been standardized on guinea-pigs and cattle very many times. He was well aware that the colostrum of tuberculous cows might contain tubercle bacilli, but unfortunately, to meet with the owners' wishes, the risk of young calves being infected with tubercle from such colostrum had to be taken. He could not, off-hand, give the contents of the medium used for the production of quadruple strength tuberculin.

The Transference of Immunity from Ewe to Lamb.

By T. DALLING, J. H. MASON, and W. S. GORDON.

DURING the course of our researches into lamb dysentery we devised a method of protecting lambs from dysentery by the inoculation of the mother sheep with a toxin-antitoxin mixture, prepared from the toxin of an anaerobe isolated from lesions in naturally infected lambs. Field results bear out that lambs are protected by such a method: thus in 1926, 2·34 per cent. of lambs born from inoculated ewes died of lamb dysentery, while 18·44 per cent. of lambs from uninoculated ewes died. The figures for 1927 are 0·87 per cent. of lambs from inoculated ewes died, while 8·04 per cent. of lambs from uninoculated ewes developed lamb dysentery.

We have carried out a series of experiments which show that the inoculation of ewes with the above mixture causes the formation of antitoxin in their serum and that lambs born from such sheep have a similar amount of antitoxin.

Method of Testing Serum for the Presence of Antitoxin.

A standard toxin was made by growing the anaerobe in meat broth for 12-15 hours, filtering through paper pulp and sand and then through a Berkefeld filter. The filtrate is precipitated with ammonium sulphate, the precipitate is dried *in vacuo* and the residue is finely powdered. A weighed amount of this powdered toxin is dissolved in saline solution when tests are to be carried out. The toxin in this powdered form has retained its toxic and other qualities for many months. Experiments were carried out to determine the minimum lethal dose of the toxin by the mouse intravenous method, and the minimum skin-reacting dose by the guinea-pig intradermic methods. The smallest amount of toxin causing the deaths of mice and causing skin reactions in guinea-pigs, was used in the testing of the antitoxic content of the sheep sera. Fatal or reacting doses or multiples of them are mixed with 0·2 c.c. of serum, the mixtures allowed to stand at room temperature for about one hour and then injected into mice intravenously or into guinea-pigs intradermically; results are read in the mouse experiments in twenty-four hours and in the guinea-pig tests forty-eight hours later.

Testing of Serum from Inoculated Sheep.

Tests of many samples of serum from inoculated sheep have been made. Table I is the result of tests carried out on samples from ten sheep which were

brought to the laboratory for inoculating and testing. These sheep were injected twice during pregnancy and were bled before injecting, after the first inoculation, and on several occasions after lambing. No antitoxin could be demonstrated in any sample obtained before injecting, while in three sheep after one injection, 0·2 c.c. serum protected against one fatal dose of toxin. Of the ten sheep, three failed to develop any detectable antitoxin after the second injection. Sheep 41 is of interest in that it showed the presence of antitoxin after one, but not after two injections. On the day it received its first injection it was necessary to administer 40 c.c. hyper-immune serum. Our test therefore detected passive immunity.

TABLE I.—ANTITOXIN IN SERUM OF INJECTED SHEEP.

Sheep	Before injection	After first injection	After second injection (in days)		
40	Nil	P. 1	7 P. 3	24 P. 3	34 P. 3
41	Nil	P. 1	17 nil	24 nil	44 nil
42	Nil	P. 1	17 P. 2	24 P. 1	44 P. 1
43	Nil	Nil	3 P. 2	21 P. 3	32 nil
44	Nil	Nil	14 nil	21 nil	33 nil
45	Nil	Nil	17 P. 3	24 P. 1	44 nil
46	Nil	Nil	14 nil	21 nil	44 nil
47	Nil	Nil	23 P. 2	28 P. 2	54 nil
48	Nil	Nil	25 P. 1	32 nil	50 nil
49	Nil	Nil	16 nil	24 nil	44 nil

Tests done in mice i. v. 0·2 c.c. serum mixed with one or more M.L. Ds. toxin and stood for about one hour before injecting.

Nil = no protection.

7 P. 3, etc. = protection against 3 M.L.Ds. toxin when test done with serum got seven days after second injection, etc.

Testing of Lamb's Serum for Antitoxin and a Comparison with Antitoxin Content of the Mother Sheep.

Again, many samples of lamb's serum have been tested for antitoxin content. Table II is the result of testing samples of serum from lambs produced by the ewes referred to in Table I. Samples of blood were drawn on the day of the lamb's birth and again when the lamb was 7-9 days old. The results of the test indicate that the antitoxin content of the lamb's serum corresponded closely to that of the mother sheep.

Our experiments do not indicate whether such antitoxin was developed by the fœtus *in utero* or whether it was transmitted to the fœtus *in utero* by the mother or via the colostrum after birth.

Experiments will be carried out during this year's lambing season to establish this point.

TABLE II.—COMPARISON MOTHER EWE AND LAMB SERUM—PRESENCE OF ANTITOXIN.

No. ewe and lambs	Lamb serum (day of birth)	Lamb serum (7-9 days old)	Ewe serum (day of birth of lambs
41	Nil	—	Nil
42	(*a*) No P. (*b*) P. 3 (*c*) P. 3	Died P. 3 P. 3	P. 2
43	(*a*) Nil (*b*) Nil	— —	P. 2
44	(*a*) Nil (*b*) Nil	— —	Nil
45	(*a*) P. 3 (*b*) P. 3	— P. 3	P. 3
46	(*a*) Nil (*b*) Nil	Nil Nil	Nil
47	(*a*) Nil (*b*) Nil	P. 2 P. 2	P. 2
48	P. 2	P. 2	P. 1

Tests done in mice i. v. 0·2 c.c. serum mixed with one or more M.L.Ds. toxin and stood for about one hour before injecting.

Nil = No protection—all mice died.

P. 3, etc. = protected mice against 3 M.L.Ds. toxin, etc.

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[*May* 23, 1928.]

A Further Contribution to the Subject of Aplastic Anæmia.

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ABSTRACT.—In the description of the condition of benign aplastic anæmia in pigs, attention is directed, among other things, to changes in the bone-marrow which seem of fundamental importance in understanding normal erythrogenesis.

It is possible that an indirect van den Bergh reaction can be converted into an immediate direct by controlling the H-ion concentration suitably, either with buffer solutions or with less dissociable acids, such as acetic, used in preparing the van den Bergh reagent. The question of the interpretation of direct and indirect reactions, therefore, would seem to be reopened.

Following Minot and Murphy's work, a pig with aplastic anæmia was fed with liver. The lesions in the liver, considered with the marked improvement in the blood which followed on liver feeding in this case, lead one to regard the condition as one of blood and marrow inefficiency due primarily to hepatic insufficiency.

A comparison of pernicious anæmia with the benign aplastic anæmia of pigs seems to indicate that in pernicious anæmia there is more than a mere functional disturbance of the marrow. The marrow appears to be fundamentally and organically affected, and this appears to be the pathological basis of the disease—a point which has already been discussed more fully elsewhere [14]. A parallel may be drawn between the varieties of aplastic anæmia, such as already exists for ordinary anæmias. We have, thus, the secondary or benign anæmias on the one hand; and, on the other, the primary or malignant anæmias, such as pernicious anæmia. Correspondingly, we have such benign or secondary aplastic anæmias as that described in the pigs; and malignant or primary aplastic anæmias such as those in benzol poisoning, etc. That aplasia should be so marked a feature in iron deficiency, as compared with other secondary anæmias, is probably due to the persisting nature of the cause.

I HAVE already discussed aplastic anæmia in its general bearings elsewhere [1]. It is therefore unnecessary to treat of it at length here. Certain aspects of a more or less benign variety—iron deficiency—will be taken for more particular treatment.

The condition has been observed and investigated in pigs. It occurs in the modern pig to a great extent, possibly because this animal has been bred for the development of rapid growth. In essence, it appears to depend on a deficiency of iron in the milk relative to the protein content, considering the excessively developed growth potential of the young animal. For reasons which need not be gone into here, it does not occur if the sows are good milkers, even in the conditions already outlined. Its onset occurs at the date of birth and reaches its climax at the third or fourth week of life. Seen at this stage the animals are abnormally fat, stocky, pale, affected with diarrhœa and with heaving or pumping breathing, from which the disease derives the name of "thumps" At this stage, profound anæmia with hæmoglobin index equal to or greater than 1 is present and sudden death is frequent. Post-mortem in such cases shows chiefly enormous dilatation of the heart, effusion into all the serous cavities, œdema of the lungs, and extensive fatty change in the centre of the liver lobules. The blood changes will be discussed later with one of the sequelæ.

At or before this stage, the disease can be prevented or its symptoms and sequelæ ameliorated, in most cases, by administering some preparation of iron. In a certain number of cases, however, sequelæ occur. These are of two types; each may occur separately, though an admixture of the two is usual. In the first form, faulty

resorption of the effused fluids causes adhesions between the opposed surfaces of the serous cavities. This produces adherent pericarditis, pleurisy, and peritonitis; the affected animals cease to grow and become hairy undersized "shargars" or wasters. The second pathological syndrome, which may arise, is the basis of this paper. Reference has been made to the anæmia and the fatty change in the centre of the liver lobules. In these cases, the latter becomes worse and the fat disappears together with the containing liver cells. Eventually, the liver lobule, enormously enlarged, is represented by a thin marginal zone of liver cells, the centre being entirely filled with replacing blood. This blood is not of the nature of a hæmorrhage for it is still within the circulation With these phenomena, the anæmia persists and is accentuated, while other changes occur which will be immediately described.

A fairly complete clinical and pathological examination has been made on at least thirty cases of this type. Though individual cases showed slight variations, these were so unimportant that a composite picture of the disease is not inaccurate. Such pigs, after the initial acute stage, appear to recover somewhat but never become quite normal. They are paler than usual and their appetite is never keen. Nevertheless, they are not undersized and hairy, like those affected with the adhesive phenomena; they grow fairly well with a clean skin, until about the age of four months. Their weight may then be 50-70 lb. as compared with the normal of 100 lb. Then the appetite suddenly fails, the sides fall in, the tips of the ears become deeply cyanosed, indicative of failing circulation, and the animal usually dies in a few days. Percussion indicates fluid in the peritoneal cavity.

At this stage the blood shows a red-cell count of about 3,000,000 per c.mm., as compared with a normal 7,000,000—8,000,000 per c.mm. The hæmoglobin registers 30—40 per cent. on the human scale as compared with a normal 80 per cent. The hæmoglobin index thus varies from just under to just over 1. A fairly marked leucocytosis is present, rising in some cases to about 50,000 per c.mm. as compared with a normal of 10,000. Blood-films show extensive anisocytosis and some slight poikilocytosis. Macroblasts and normoblasts are numerous. Megaloblasts are not present: polychromasia is marked. The significance of these findings will be discussed later.

A jaundiced condition of the skin, etc., absent for the greater part of the duration of the disease, is common during the last few days of life, when the circulation is failing. In harmony with this, a direct or indirect van den Bergh reaction is absent during the earlier period. Curiously enough, however, the blood-serum, as well as the effusions (which are of a yellow tint), during the jaundiced period give only an indirect reaction with the ordinary van den Bergh reagent. Nevertheless, the jaundice is undoubtedly obstructive, Iron pigment is entirely absent, let alone a hæmo-siderotic condition in the organs, such as would occur in a hæmolytic jaundice. At the same time, sections of the liver show an accumulation of yellow pigment in the centre of the liver cell columns near the commencement of the bile ducts. This yellow pigment gives a positive reaction with van den Bergh's reagent when tested microchemically, indicating that it is of the nature of bilirubin. Its position indicates that it is being dammed back and points to obstruction.

From this it would seem that the last word has not been said on the clinical significance of the van den Bergh reaction. If, following Davies and Dodds [2] in a suggestion regarding the importance of H-ion concentration in the reaction, the van den Bergh test be made with serum or effusion fluid of affected pigs, diluted in buffer solution of pH = 7, instead of in distilled water, an immediate direct reaction is given. The same result is obtained, even when distilled water is the diluent, if a less dissociable acid than HCl—acetic acid—in the same percentage strength is used

in making up the No. 1 solution of the van den Bergh reagent. It seems, therefore —as Davies and Dodds point out—that for the van den Bergh colour change, a certain pH is necessary. That the "indirect" reaction succeeds where the direct fails may be explained on the basis that alcohol lessens the dissociation of the HCl of the ordinary reagent and so raises or keeps the pH to a level more favourable for the colour change. Adler and Strauss' [3] finding as to the beneficial effect of Caffeine-sodium—salicylate in hastening the reaction may have a related explanation (hydrolysis and increase of pH). Thannhauser and Andersen's [4] modification with alcohol and ammonium sulphate can also be explained by decreased dissociation. Bilirubin itself, being acidic in character, will also affect the pH.

It would seem, therefore, that unless due regard be had to the H-ion concentration, a really obstructive case of jaundice may give an indirect reaction, and the same substance, bilirubin, can give two different reactions—the direct and the indirect. Ordinarily, the direct and the indirect reactions are interpreted as produced by two different substances and as indicative of two different clinical conditions. This deduction, in the circumstances, is untenable unless one clinical condition as its essence supplies the milieu for the direct reaction, while another supplies that for the indirect, the basal substance, bilirubin, remaining the same in both cases. The following evidence is against this.

The experiments recorded here deal with a condition of obstructive jaundice in pigs, where, by suitable H-ion concentration, an initial indirect reaction is changed into an immediate direct. The amount of bilirubin in the serum of the pigs concerned was relatively small and this might account for no immediate direct reaction being observed with ordinary van den Bergh technique. This being kept in view, a sample of serum was obtained from a case of marked obstructive jaundice in a human being. It was deeply yellow and was tested within a few hours of withdrawal. With the ordinary van den Bergh technique (HCl reagent, distilled water dilution), it gave the merest trace of a pink reaction in one minute; with the indirect-alcohol, or dilution with buffer solution, it gave an instantaneous deep carmine with the HCl van den Bergh reagent; with the acetic acid reagent it gave a similar instantaneous reaction whether the dilution was with distilled water, buffer solution, or alcohol. If, therefore, the indirect reaction in colour change intensity is indicative of the amount of bilirubin present, then, in this case, the direct reaction, while giving a faint qualitative indication of its presence, is unreliable as to the amount. It might even be that, if only slightly less bilirubin had been present in the serum, a negative immediate direct reaction would have been recorded. Here it may be noted that the routine estimation of the amount of bilirubin in serum has to be made by the indirect method. The time taken for the appearance of the colour change, depending largely on the pH, seems to be the chief distinction between the immediate direct and the delayed direct or indirect. There is, however, another time-conditioned phenomenon in the reaction. This is the change of the pink colour to a deep yellow, which appears also to depend on the pH. The indirect estimation, by its sudden arrival at a maximum, cuts out errors due to these two variables.

It seems, therefore, that a not sufficiently stressed factor in the appearance of the van den Bergh direct colour reaction is the amount of bilirubin present. Hence, a large quantity of bilirubin in a serum may make up by mass action for an incorrect pH in regard to the appearance of some degree of colour reaction. No such compensation is possible in the body fluids when the bilirubin is small in amount. For lack of a sample, I have been unable to test whether the serum in cases such as pernicious anæmia, when suitably buffered or when tested with acetic van den Bergh reagent, will give an immediate direct reaction.

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Davies and Dodds [loc. cit.], while pointing out the importance of H-ion concentration and of buffering solutions in the colour reaction, did not apply these findings to explain the difference between the direct and indirect reactions. They supposed that the latter was due to the bilirubin being transformed into biliverdin, a view which one cannot corroborate.

Van den Bergh [5] noted cases where, in terminal heart failure, the blood-serum gave an immediate direct reaction, without ascertainable appropriate lesion in the liver. Such cases seem comparable with those of the jaundiced pigs described here. In the pigs, however, the story is more easily read, for, in addition to the terminal heart failure, visible for a few days before death, we have profound changes in the liver, which would predispose to bile obstruction, demonstrated microscopically. The obstruction, or at least the jaundice, did not appear, however, until the heart began to fail.

We may now return to a consideration of the changes in the blood and blood-forming organs. The blood findings have already been discussed in the general statement. With regard to the marrow, the bones of the limbs and axial skeleton, excepting the lower end of the tibia (radius and ulna not examined), were filled with deep red marrow, reminding one of the conditions in pernicious anæmia. The shafts of the long bones contained marrow without supporting cancellous tissue. In the tibia there was a clear line of demarcation between the advancing red and white fatty marrow (fig. 12). For the time being we will concentrate on an examination of this junction. Sections of this area showed that it was one of intensive preparation for erythropoiesis. Between the abundant juxtaposed fat cells are the reticular elements giving rise to hæmacytoblasts (fig. 1); these enlarged to become megacaryocytes (fig. 2), which subsequently broke up amitotically to form prenormoblasts[1] (figs. 3, 4, 5). The prenormoblasts retained at first in their cytoplasm the eosin tinge (Leishman's stain) of the cytoplasm of the megacaryocyte. Later, this changed to the golden yellow of the mature erythrocyte while the nucleus disappeared (fig. 6). Mitotic phenomena in the prenormoblastic and normoblastic cells were absent, the whole production of erythrocytes apparently taking place from the breaking up of the megacaryocytes.

These changes took place inside the blood-vessels (figs. 1, 2, 3, 4, 5, 6), and corroborate views expressed by me elsewhere [6]. From experiments on pigeons and rabbits, Doan, Cunningham and Sabin [7] have come to somewhat similar conclusions regarding the intravascular nature of erythropoiesis, but with a distinct difference. They regard the erythropoiesis as taking place in already existent but collapsed inter-sinusoidal capillaries. It is difficult to see how this can be, as it premises the existence in pure, fatty marrow which had never functioned before, of a network of capillaries fine enough to surround each individual fat cell. There is no need for such supposition, and the view held here is that, as in the embryo, the primitive mesenchyne or reticular cell, lying dormant for the time being between the fat cells, forms at once the stem cells of the red blood-cells, and their enclosing capillaries, which latter subsequently join up to the existent circulation.

In this advancing edge of the red marrow, as already mentioned, erythropoiesis is the dominant activity. In the already formed red marrow, it still plays a major rôle, as evidenced by the number of megacaryocytes, fully formed or breaking up into accumulations of prenormoblasts and normoblasts (figs. 12, 13 and 14). In this area, fully formed erythrocytes are rare. Here and there, one finds small accumulations of myeloblasts and myelocytes. It is significant, however, that hæmocytoblasts or lymphoidocytes are relatively few compared with the condition in pernicious anæmia. Fat cells, of course, are much reduced in number.

[1] "Prenormoblast" includes premacroblast and prenormoblast.

In the ribs, appearances corroborate and amplify those already described. One finds, for instance, conditions such as those mentioned for the red marrow of the tibia (fig. 15). Such hyperplastic areas, however, may be continuous with and pass insensibly into other large areas which are apparently ripening areas and where the cells are represented almost entirely by ripe non-nucleated erythrocytes (fig. 16). In their turn, these areas may give place to others, where, apparently, the ripened erythrocytes have passed into the general circulation, leaving the marrow in a denuded, aplastic condition and apparently without the capacity to form new blood-cells (fig. 17).

The appearances in these marrows were found with slight modifications in the marrows of young pigs affected with the acute disease at three weeks of age. In these pigs, however, the medulla of the long bones was filled with pale pink cancellous tissue and aplasia was more in evidence. In both, however, the essential change appears to be a perverted and excessive hyperplasia of erythropoietic tissues, resulting in exhaustion and aplasia. The urge to the hyperplastic changes in both cases is apparently a diminished oxygen-carrying capacity of the blood, due to lack of hæmoglobin-forming materials.

In the young pigs at three weeks, the administration of iron, if carried out efficiently in not too severe cases, usually produces entire recession of the pathological phenomena. If these conditions were not fulfilled, cases resulted such as form the basis of this communication. In these, administration of iron produced no beneficial result and, if the conditions were at all severe, the issue was almost always fatal. Elsewhere [8] and before I was aware of Minot and Murphy's [9] beneficial results of feeding liver to pernicious anæmia cases, in the discussion of aplastic anæmia, I concluded from the evidence that the functions of liver and bone-marrow in red blood-cell formation are complementary, and that the liver elaborates iron-containing phospho-lipoids which it passes on to the marrow for the construction of red blood-cells. At the time, it seemed impossible to obtain direct proof of this, but Minot and Murphy's results led one to reconsider the situation. It then appeared that this pig condition provided, already prepared, the inactivation of the major portion of the liver tissue by a simple, bland and almost perfect experimental procedure. Briefly, in this case a non-drastic procedure (lack of iron), no longer operative at the time of observation, has caused the major part of the functioning liver tissue to be replaced by blood still in circulation. It therefore seemed a good opportunity to test the efficacy of administration of liver since iron had failed. The scheme was difficult to carry out; before blood examination, etc., proved an animal to be in a sufficiently advanced stage to put the remedies to as severe a test as possible, the appetite had entirely ceased and the animal was often on the point of death—several animals died before anything could be done. Forced feeding was then resorted to with more success and the relative protocol is as follows:

Pig 580: weight 79 lb.: born August 21, 1927.

February 20, 1928; hæmoglobin 40 per cent., red blood-cells 3,750,000 per c.mm. Blood-films showed the characteristic changes already described.

February 23, 1928; hæmoglobin 40 per cent., red blood-cells 3,150,000 per c.mm., white blood-cells 42,000 per c.mm.

February 27, 1928; hæmoglobin 40 per cent., red blood-cells 3,800,000, white blood-cells 16,000 per c.mm.

No jaundice shown; blueness at the tips of the ears; the pig was emaciated with sunk flanks, was entirely off its food and had to be forcibly fed. From this time onward it was given by the mouth, twice a day, a pint of warm milk mixed with a half-pound of minced ox liver and a drachm of tincture of digitalis.

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February 28, 1928; blood was drawn; the serum was clear and colourless, and van den Bergh's reaction negative.

March 1, 1928; the pig was noted by independent and expert observers to be much better; improvement was visible also on March 2, 1928, and March 3, 1928.

March 4, 1928; the pig was worse again.

March 5, 1928; the blood was examined; hæmoglobin was now 80 per cent. or normal; red blood-cells 7,500,000 per c.mm. or normal; white blood-cells 41,500 per c.mm.; blood films showed very marked reduction of the anisocytosis and polychromasia.

March 6, 1928; the pig was found dead. Death was probably due to aspirational pneumonia (found post mortem) caused by the forced feeding. There was no jaundice, and no effusions into serous cavities. The liver was much the least affected of any in the series of cases, although it bore traces of the disease. The bone-marrow conformed to the appearances in the other cases. Though it was difficult to be sure, there seemed to be evidences microscopically of an attempt to return to normality in that fat cells and ripe erythocytes appeared to be more abundant.

Points to be noted in this case are the marked initial improvement clinically, and the very decided improvement in the condition of the blood. An assessment of the changes in the liver is, however, difficult. It was impossible, of course, to know its condition at the commencement of the treatment; again, this pig was older than the usual subject, thus affording a longer opportunity for spontaneous betterment. It seems unlikely that the nature of the lesion would allow of any marked improvement in the liver in a week's feeding on liver.

We may now compare this condition with pernicious anæmia of human beings. As resemblances we have, in the early cases, the adiposity of the body, and the fatty change in the centre of the lobules of the liver. Several pig cases showed an entire lack of acidity of the stomach contents, while in both conditions there is a tendency to dropsy and accumulation of fluid in the serous cavities.

Superficially, the blood conditions appeared similar, with anisocytosis and poikilocytosis, especially in pig cases with hæmoglobin index greater than 1. In the pig condition, however, the large erythrocytes are macrocytes, not megalocytes, while the large erythroblasts are macroblasts, not megaloblasts. Again, polychromasia is a marked feature in the pigs, it being quite otherwise in pernicious anæmia. All these changes in the pigs' blood indicate a vitally active and not a decadent marrow, though, for the time being, its functioning is aberrant.

The leucocytosis in the pigs' blood, again, differentiates it from that of pernicious anæmia, as does also the absence of hæmosiderosis.

Redness of the whole marrow in the pigs supplied again a superficial resemblance to the condition in pernicious anæmia. Microscopic examination showed, however, marked differences. In the pig marrow the predominant feature is the increase in the number of megacaryocytes and their splitting up to form macroblasts, normoblasts and erythroblasts. These in their turn form the corresponding erythrocytes. In pernicious anæmia, however, as has been discussed elsewhere [10], the characteristic is the paucity of megacaryocytes, the abundance of lymphoidocytes or hæmocytoblasts and the fewness of the cells of the type of erythroblasts (Ellermann [11], Helly [12]). Peabody [13] describes similar conditions if one allows for the fact that his "megaloblasts" should be termed, according to the usual nomenclature, hæmocytoblasts or lymphoidocytes.

In the pig disease, the condition of the blood and marrow seems without doubt to be referable directly to hepatic insufficiency. The pathogenesis of pernicious anæmia has been discussed elsewhere [14]. The findings in the present instance tend to support the views expressed there. Considering the incomparably greater

damage to the liver in the pig, the comparable amelioration on liver feeding in both conditions, and the essentially different histological appearances in the two marrows, it seems that, in pernicious anæmia, the assumption of some basic organic change in the marrow in addition to possible liver changes is justifiable. The appearances in the pigs' marrow may be assumed to be those due to simple gross liver damage which excludes it from functioning. There is something more than this in pernicious anæmia. In it, there is a return along the basal stem cell lineage to the condition of the marrow and blood in the embryo. To cause this there must be something organically wrong with the marrow which contributes to the essential malignancy and perniciousness of the condition. The possible causes and consequences of this condition of affairs have been discussed elsewhere [14] and need not be entered into here. However, malignant aplastic anæmias, such as occur in T.N.T. and benzol poisoning, etc., and which are of quite a different order from the benign aplastic anæmia of the pigs, supply a possible clue. Here, a specific poison has fulminantly damaged the marrow beyond the possibility of reactional hyperplasia, a condition of profound aplasia being at once established. In pernicious anæmia the damaging agent seems to be not so forcible and destructive, and to allow of a period of hyperplasia, albeit advanced and depraved, before final aplasia sets in.

Peabody (loc. cit.) has suggested that the feeding of liver in pernicious anæmia cases acts by a process of maturation or ripening of the forerunners of the red blood-cells. While this may be part of its action in iron deficiency and in pernicious anæmia, it cannot be its whole activity in this last disease, for in it the betterment consists in a change from the embryonic to adult blood formation. Such a transformation, from the primitive to the definitive blood type, in the embryo is associated with the onset of hepatic erythrogenesis. Both occurrences, therefore, seem to point to the existence of some deep-seated biological relationship, other than ripening, between the two tissues. Incidentally, no proof exists that megaloblasts (in the ordinary sense) mature into normoblasts and normocytes, their terminal stage being the megalocyte.

I wish to express my great indebtedness to Miss A. E. Glennie, B.Sc., for the coloured drawing of the marrow conditions; and to Dr. Lyall, Aberdeen Royal Infirmary, for a supply of obstructive jaundice serum.

I have to record my gratitude to the Carnegie Trustees for the Universities of Scotland, who have assisted me with a grant in aid of the coloured illustration.

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DESCRIPTION OF ILLUSTRATIONS.

[The first six legends refer to figures on coloured plate].

Fig. 1.—Commencing activity in fatty marrow, showing intercellular hæmocytoblasts, with pale nucleus and deep blue cytoplasm, derived from the interstitial reticular cells; these eventually form the blood-vessels and red blood-cells. Leishman's stain. See also Fig. 7. (× 380.)

Fig. 2.—More advanced stage of Fig. 1, showing megacaryocytes, derived from hæmocytoblasts, inside blood-vessel. Leishman's stain. See also Fig. 8. (× 380.)

Fig. 3.—More advanced condition, showing several large ripe megacaryocytes inside blood-vessel, some of them breaking down into prenormoblasts; note the eosinophil tinge of the cytoplasm of the megacaryocytes and prenormoblasts. Leishman's stain. See also Fig. 9. (× 380.)

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Fig. 4.—More advanced condition, showing several large ripe megacaryocytes, broken up into prenormoblasts inside a vessel; note the eosinophil tinge of the cytoplasm of the prenormoblasts and their angular contour. Leishman's stain. See also Fig. 10. (× 380.)

Fig. 5.—Further stage, showing collection of prenormoblasts from broken-up megacaryocytes, some of prenormoblasts changed into normoblasts with orange-coloured cytoplasm. Leishman's stain. See also Fig. 11. (× 380.)

Fig. 6.—Further stage, showing still further formation of normoblasts and erythrocytes. Leishman's stain.

Fig. 12.—Low-power view of marrow junction in tibia of pig 585, showing the red marrow gradually invading the white fatty marrow. Note numerous megacaryocytes. Figs. 1-11 were obtained in this area (× 90.)

Fig. 13.—Low-power view of hyperplased red marrow, showing few fat cells and cellularity, the denser areas of which consist of megacaryocytes or accumulations of erythroblasts. (× 90.)

Fig. 14.—Same under oil immersion, showing megacaryocytes, breaking up into prenormoblasts, clumps of prenormoblasts and normoblasts, and a few hæmocytoblasts. (× 450.)

Fig. 15.—Rib marrow, showing much the same condition as in Fig. 14; note the absence of erythrocytes. (× 300.)

Fig. 16.—Rib marrow, adjoining area to that of Fig. 15; note the numerous fully-formed erythrocytes, and the paucity of cells of the type of megacarocytes and erythroblasts. (× 300.)

Fig. 17.—Rib marrow, adjoining area to those in Figs. 15 and 16; note the almost entire disappearance of erythroblasts and erythrocytes: the ripe erythrocytes have apparently escaped into the circulation, leaving the marrow in a torpid aplastic condition. (× 300.)

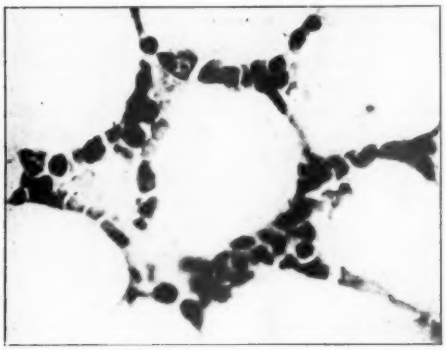


Fig. 7.

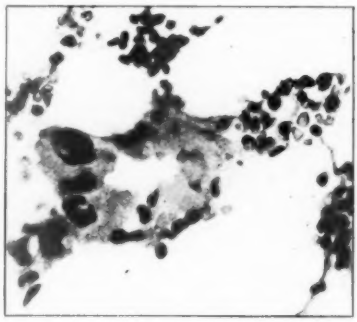
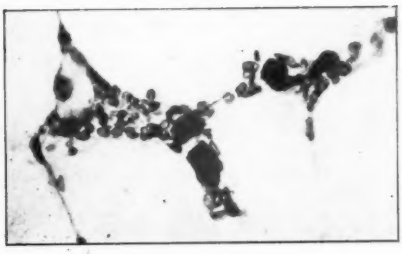


Fig. 9



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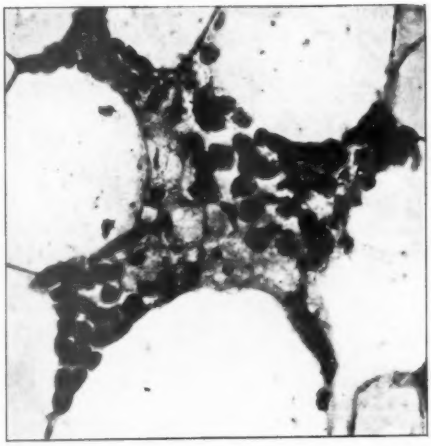

Fig. 10.

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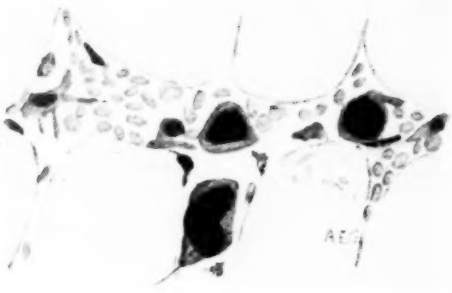

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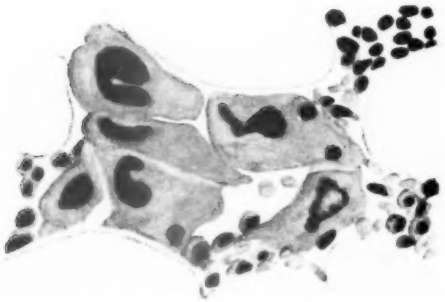

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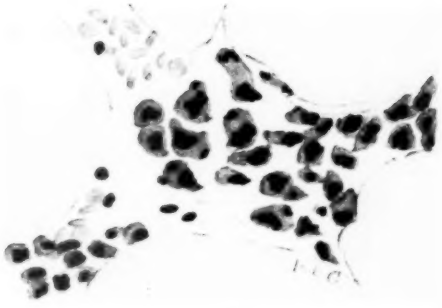

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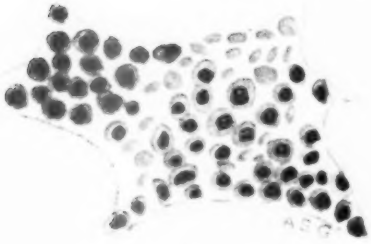

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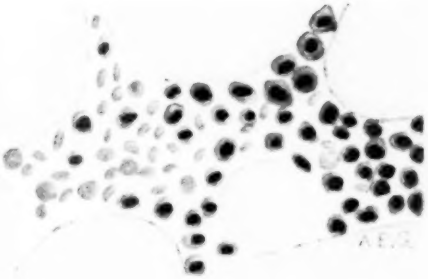

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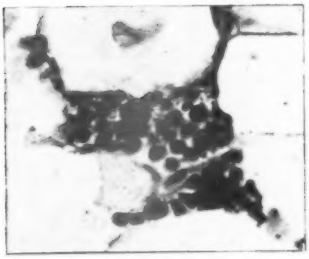
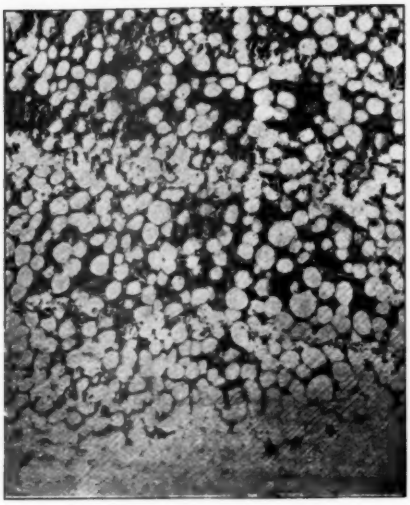


FIG. 11



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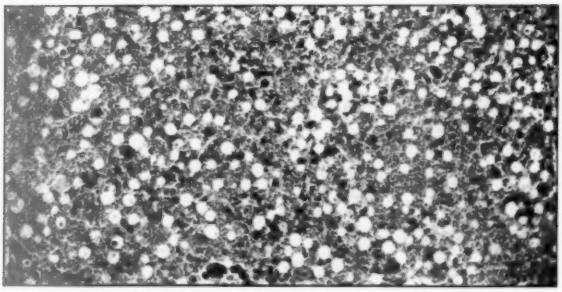

FIG. 13.

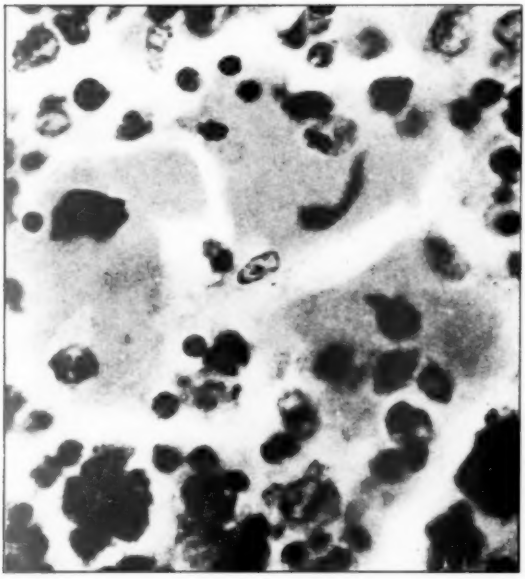

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FIG. 14.

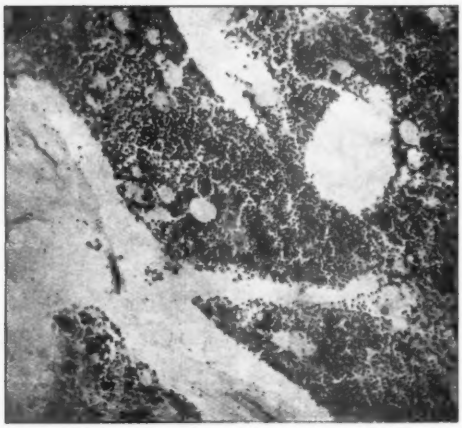

FIG. 15

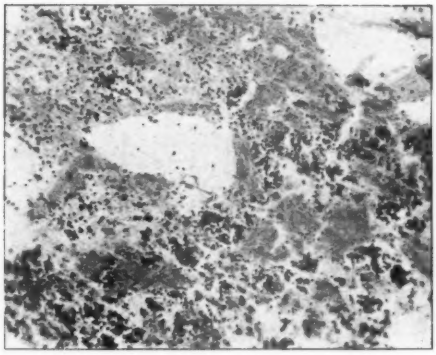

FIG. 13.

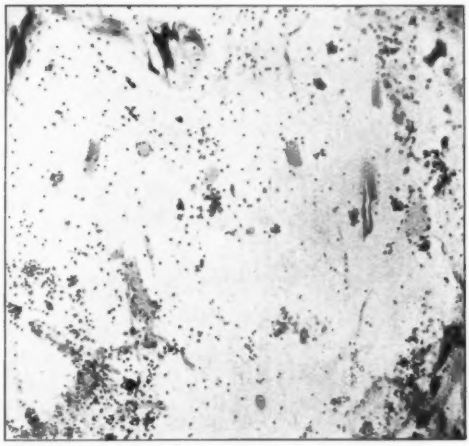

FIG. 17.

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Discussion.—Dr. H. P. BAYON: The paper, slides and specimens clearly demonstrate that this peculiar anæmia in pigs is accompanied by an active proliferation of the red bone-marrow, followed by lesions in the liver. The aplasia of the marrow appears to develop late in the course of the disease, being a terminal symptom or condition.

The morbid alterations of the spleen are not unimportant, for they may shed light on one of the functions of this organ. Many years ago, von Koelliker said that the spleen was a graveyard for erythrocytes. In the course of observations on various animals suffering with different types of anæmia, I have come to the conclusion that the spleen destroys only immature or altered erythrocytes.

I would suggest that this disease in pigs belongs to a group of anæmias, of which the principal feature is a primary hyperplastic condition of the marrow followed by aplasia.

Dr. A. PINEY: The term "aplastic anæmia" should be confined to conditions in which anæmia develops because the bone-marrow has become aplastic. An anæmia associated with hyperplastic marrow and evidences of regeneration in the peripheral blood should not be called aplastic anæmia even if, in the later stages, the marrow becomes atrophic. An origin of erythrocytes from megakaryocytes is difficult to accept, in view of the fact that the number of megakaryocytes can be enormously decreased by careful administration of benzol without, at the same time, causing anæmia. Further, it is difficult to account for the disappearance of the characteristic azurophilic granules from the megakaryocytes during their conversion into red corpuscles.

Dr. KNYVETT GORDON: I agree that it is difficult to see why this disease should have been described as "aplastic" anæmia as it is only in the terminal stage that aplasia supervenes.

In the human subject, aplastic anæmia is associated with a definite blood picture so well described by Piney in his "Recent Advances in Hæmatology." The condition is characterized by a great reduction in red cells, white cells and platelets, together with absence of anisocytosis, polychromasia and nucleated red cells, the differential count showing only a relative predominance of lymphocytes. This does not resemble the blood picture of Dr. McGowan's pigs. Both from the clinical and from the pathological point of view I emphasize that the disease in the human subject has no relation to pernicious anæmia. Aplasia of the hæmopoietic system is a late condition due to exhaustion of the bone-marrow from a variety of causes of which, in my experience, septic infection is the most common. An example can, however, be seen in the terminal stages of myeloid or lymphatic leukæmia when either has been treated by excessive doses of X-rays or benzol. I have met many instances of this, and it is a warning against the use of X-rays uncontrolled by frequent blood examination. It is a great mistake to attempt to reduce the blood-count by these methods, for the majority of sufferers from leukæmia are most comfortable when the count is fairly high.

Apart from leukæmia, aplastic anæmia is usually curable by detection and removal of the cause, vaccine therapy being of great utility in the septic cases, combined with complete rest, a generous diet and a supply of the essential vitamins, especially vitamin D. I have found neither iron nor arsenic of use when the cause has not been dealt with.

Dr. F. PARKES WEBER said he would not touch on the questions on which Dr. Piney and Dr. Knyvett Gordon had already spoken. The excellent microscopic sections shown by Dr. McGowan from the livers of the diseased pigs (in a late stage of the disease) suggested to him (Dr. Weber) an advanced stage of centro-acinous atrophy, similar in nature to the microscopical change not very rarely met with at post-mortem examinations in various toxic and septic cases in human beings. A somewhat similar change constituted the "nutmeg liver," found at the necropsy on cardiac cases with passive venous congestion, especially when death was long delayed, and when there was a terminal infection of some kind. The positive van den Bergh reaction for bilirubin in the blood-serum would be at least partly of the indirect type.

He would suggest experimental splenectomy in the diseased pigs at a relatively early stage of the disease, and also the trial of one of the iron-free and protein-free liver-extracts that had been used with such wonderful success in human cases of pernicious anæmia. He thought that the therapeutic results (if any) obtained by these experiments might help to throw further light on the nature and ætiology of the disease in pigs under discussion. In

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human beings splenectomy was often clinically curative in cases of congenital or familial hæmolytic (acholuric) jaundice. In the latter disease removal of the spleen removed the anæmia by preventing undue hæmolysis of the specifically defective, i.e., "fragile" erythrocytes. It now seemed that in pernicious (Addisonian) anæmia the excessive hæmolysis was also chiefly of the cells badly turned out by the bone-marrow, the curative action of the iron-free and protein-free extract of liver being due to a stimulation of the bone-marrow, causing it to produce less defective and numerically more erythrocytes, which were not so easily hæmolysed in the spleen and elsewhere.

Dr. GEORGE SCOTT WILLIAMSON: Any attempt to approach the problem of pernicious anæmia through direct experiment is to be welcomed, and I cannot see the import of the criticisms of Dr. Gordon and Dr. Piney.

Dr. McGowan seems to have succeeded in inducing a definite progressive aplasia of the erythroblastic tissue in the marrow. This is accompanied by evident reactionary hyperplasis which is focal and not general and to be looked upon as the limit of compensation possible at this stage in his animals. The compensating effort in the liver is apparently very striking and peculiar, but it is the persistent iron deficiency that links the marrow and the liver in some complementary cycle. In these experiments the iron deficiency is absolute and direct, in pernicious anæmia it is a secondary deficiency not absolute but relative, since there is a store of iron in the tissues of many organs. It is significant that liver feeding seems to answer as a therapeutic agent both in the experimental aplastic form and in the pernicious form.

Dr. McGowan's study of the van den Bergh reaction will have a direct bearing on the clinical use of this test.

Dr. MACGOWAN (in reply) said: The function of the bone-marrow is to produce normal circulating blood. Any *true* hyperplasia of it should accomplish this within very slight limits. In spite, however, of the great increase in quantity of active marrow (of a type) in the present cases, the circulating blood is entirely and markedly abnormal, in both the quality and the quantity of its cellular elements; in spite of the "hyperplasia," blood is *not* being formed. It would seem, therefore, that there is every justification for designating the condition an aplastic anæmia. In the marrow itself again, the aberrant hyperplasia in time gives place, without fail, to an aplasia. It is no argument against naming the present condition an aplastic anæmia to urge that true aplastic anæmia has no hyperplastic stage of the marrow. In such cases the damaging agent has been so intense that the marrow is killed outright, and any reactive hyperplasia has been suppressed. Ordinary pernicious anæmia, aplastic pernicious anæmia, and ordinary aplastic anæmia, illustrate the stages suggested as occurring in the marrow.

The part played by the spleen in this disease is not important. In some cases—suckling pigs with the disease—a certain amount of phagocytosis of red blood-cells was observable in cells of the splenic pulp. The circulating red blood-cells in such cases, however, are abnormal, and elsewhere I have suggested that, in pernicious anæmia, this abnormality of the red blood-cells leads to their hæmolysis (and consequent hæmosiderosis), and not the hæmolysis that produces the conditions that give rise to the aberrant cells of the blood. Any hæmolysis in the pig condition, however, is slight, because there is an entire absence of hæmosiderosis in the organs. Apart from this the disease has a definite concrete pathogenesis of another kind.

With regard to Dr. Piney's objections to the megacaryocyte producing erythrocytes, the illustrations which accompany the paper speak for themselves.

Dr. Knyvett Gordon thinks that, in the human subject, aplastic anæmia has no relation to pernicious anæmia. Apart from the fact that an aplastic type of pernicious anæmia is well known to exist, many authors have drawn attention to the close resemblance between toxic aplastic anæmia and pernicious anæmia, both clinically and pathologically.

The changes in the marrow, blood and liver are indissociable items in a single pathological syndrome, thus differing from the centro-acinous atrophy of the liver mentioned by Dr. Parkes Weber. As the disease has not hæmolysis as its ætiological basis, but the absence of iron in the food, it would seem that there is not the same call for splenectomy, as a curative measure, as in congenital acholuric jaundice in human beings.

Section of Epidemiology and State Medicine.

President—Dr. S. Monckton Copeman, F.R.S.

[*June* 22, 1928.]

Some Observations on Naso-pharyngeal Epidemics in Public Schools.

By J. A. Glover, O.B.E., M.D., M.R.C.P.

Abstract.—Over 80 per cent. of school sickness, whether judged by the number of cases or by the time lost, is transmitted by "droplet" infection.

The alleged increase in sickness in public schools is partly apparent, due to increased attention to minor febricula and partly real, due (1) to increased influenza prevalence, the aftermath of the great epidemic of 1918, and (2) to the increased demand for public school education leading to pressure upon accommodation, and especially to overcrowding in dormitories.

The bulk of the droplet infections are accounted for by (1) influenza; (2) feverish cold, chill, or P.U.O.; (3) tonsillitis; regular infectious diseases make a comparatively small showing. The incidence and bacteriological findings and the variations in the incidence of pneumonia and otitis media are discussed. Are these complications really secondary epidemics? Tonsillitis, bacteriological findings, milk.

Prophylaxis.—Efficacy of vaccines uncertain. Some evidence that they may diminish the onset of complications. If given vaccines should be administered before the danger period, i.e., not later than November.

Intensive prophylaxia other than vaccines during the first half of the Lent term would probably amply repay any trouble. It should include:—

(*a*) Special efforts to prevent boys returning to school after the Christmas holidays infected with influenza or febricula.

(*b*) Temperature taking for three weeks.

(*c*) Immediate isolation of all pyrexias and catarrhs.

(*d*) No work before breakfast for at least the first six weeks of the term.

(*e*) All hot baths and showers taken during the day or after games to be followed by cold showers.

(*f*) Prevention of chill in watching games, etc.

(*g*) Increased provision for drying clothes, uniforms and boots.

Infection mainly takes place in sleeping quarters, and proper spacing out of beds and thorough "cross" ventilation in dormitories is of paramount importance; instances of cross infection due to proximity of beds; illustrated by bed charts and bacteriological findings; standards of wall space, floor space and cubic space, laid down by the Royal Commission, Board of Education.

No school authority has done its duty to its pupils unless it has provided dormitory accommodation allowing at least 3 ft. of clear space between the edges of beds, and thorough and through ventilation. Until these essential wants are met, the provision of properly sited, amply spaced and "cross" ventilated dormitories should take precedence of all other building requirements.

Milk should be pasteurized.

Certain semi-isolated communities, which are under strict control, seem to be designed for epidemiological study, and of these the Public Schools of England offer an unique field for observation. Few communities are more comparable and homogeneous in the age distribution, in the nationality, and in the nurture of the individuals comprising them. Yet, with the exception of the classical work of Clement Dukes, little systematic epidemiological work seems to have been carried out with this matchless material.

Surgeon Commander Dudley has, however, shown what can be done with somewhat similar material, and his two reports[1] on the Royal Naval School at Greenwich, based largely upon the careful records of Surgeon Captain P. M. May, R.N., and

1 *Medical Research Council, Special Report Series*, Nos. 75 and 111.

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published by the Medical Research Council are, I think, even more important contributions to school epidemiology than Dr. Dukes' studies at Rugby.

As early as 1905 Dukes showed that the public schools were suffering an increased number of infectious diseases; this was largely due to the greater care exercised at home and at preparatory schools in preventing infectious disease, and to the consequent greater proportion of boys who reached public schools unprotected by previous attack. There is, I think, a general impression that a further post-war

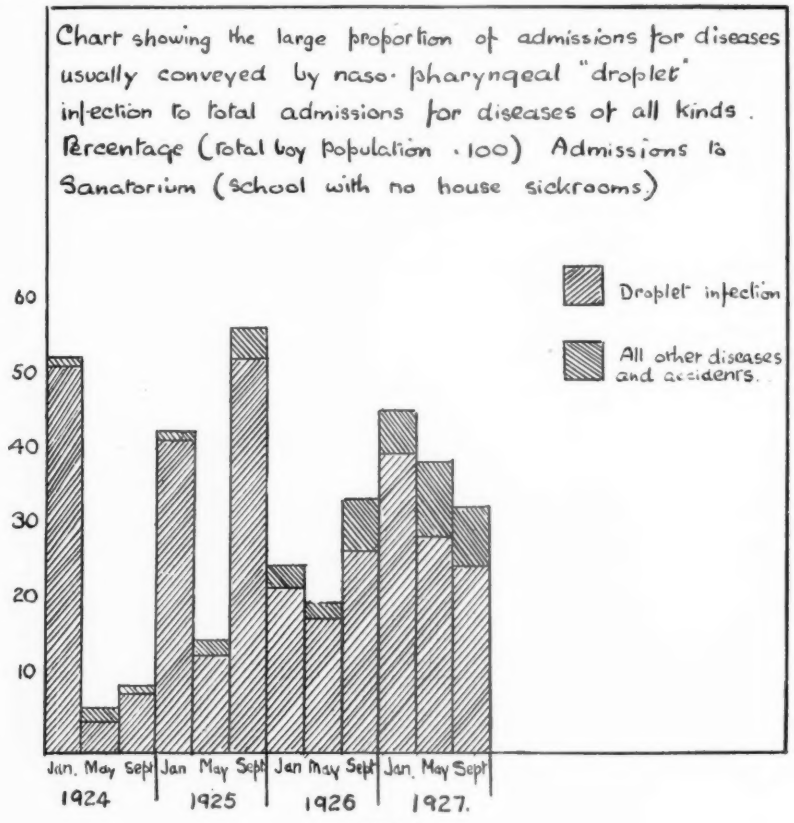
Chart showing the large proportion of admissions for diseases usually conveyed by naso-pharyngeal "droplet" infection to total admissions for diseases of all kinds. Percentage (total boy population ·100) Admissions to Sanatorium (school with no house sickrooms)
Droplet infection
All other diseases and accidents.
60
50
40
30
20
10
Jan. May Sept Jan May Sept Jan May Sept Jan May Sept
1924
1925
1926
1927.

FIG. 1.

increase has taken place. Other factors may be concerned in this post-war increase such as:—

(1) The greater prevalence of influenza since the epidemic of 1918.

(2) The great increase in the number of boys sent to public schools since the War, causing overcrowding in class-rooms and dormitories; and perhaps—

(3) The greater attention paid to what we may term (with Dr. Simey) "febricula."

I think all agree that most of the infectious diseases now prevalent in public schools are spread by naso-pharyngeal or "droplet" infection, but I doubt if we all

realize the enormous proportion of the total illness caused by direct nose-to-nose and mouth-to-mouth infection. The diagram on p. 96 (fig. 1) shows the number of boys admitted to the sanatorium each term as a percentage of the number of boys in a certain public school which I shall call "X."[1] This school is convenient for this purpose as there are no house sick-rooms, all boys ill enough to go to bed

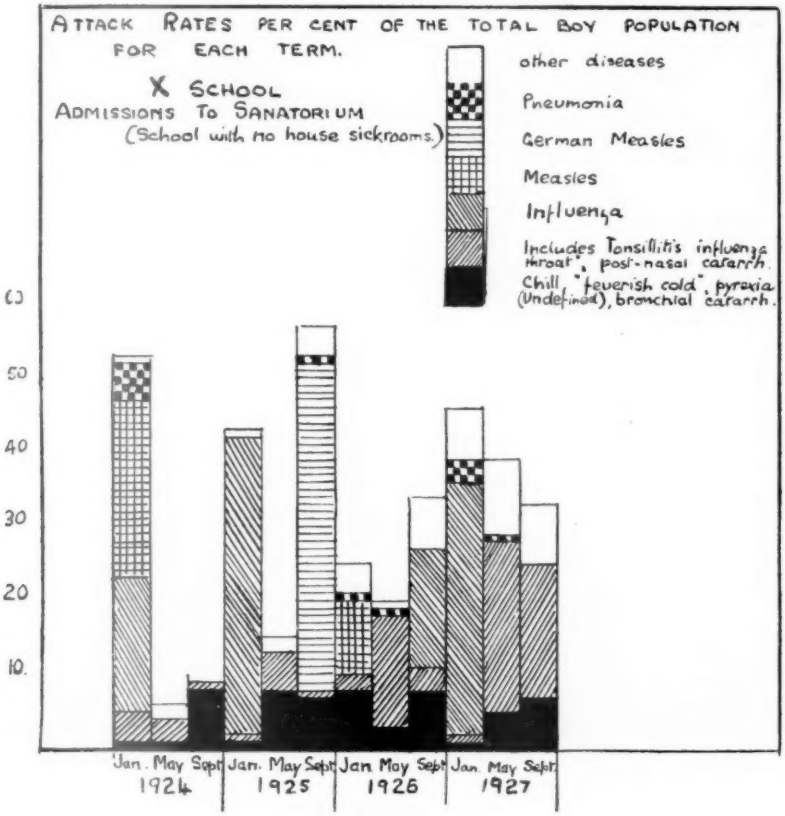

FIG. 2.

being admitted direct to the sanatorium; this fact explains the somewhat high admission rates in this case.

Let us now divide the "droplet" infection into the several diseases, as has been done by the next diagram (fig. 2). The figures are shown in Table I.

[1] Most of my information is confidential; the names of schools are given only when statistics are quoted from published work. I am thus unable to give the names of the many ladies and gentlemen to whom I am indebted for information, but whose help I very gratefully thus acknowledge.

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TABLE 1.—"X" SCHOOL "DROPLET" INFECTIONS.
Admissions Per Cent. of the Total Boys Per Term to the Sanatorium: School without House Sick-Rooms.

Year and term	Total admissions	Due to other diseases and accidents (non-respiratory)	Total due to droplet infections	Droplet infections					
				Feverish cold P.U.O. chill	Tonsillitis, influenzal throat, post nasal catarrh	Influenza	Measles	German measles	Pneumonia
1924									
1	53	1	52	1	4	18	24	—	5
2	6	2	4	1	3	—	—	—	—
3	9	—	9	8	1	—	—	—	—
1925									
1	43	1	42	1	1	40	—	—	—
2	15	2	13	8	5	—	—	—	—
3	57	4	53	7	1	—	—	44	1
1926									
1	25	4	21	8	2	—	10	—	1
2	20	1	19	3	15	—	—	—	1
3	34	7	27	8	3	16	—	—	—
1927									
1	46	7	39	1	1	34	—	—	3
2	39	10	29	5	23	—	—	—	1
3	33	8	25	7	18	—	—	—	—

On the whole, the ordinary infectious diseases of childhood occur less often than would be anticipated, although measles comes twice in the 4 years, whilst German measles comes only once, diphtheria is absent, and mumps, chickenpox and scarlet fever are absent save for one or two sporadic cases. The absence of scarlet fever is remarkable in view of the fact that the tonsillitis at this school has been shown bacteriologically to be due mostly to infection by hæmolytic streptococci often of scarlatinal type.

Influenza on the other hand occurs in epidemic form in three out of four years in the Lent terms, and once as "gastric" influenza in a Christmas term. For comparison I show a diagram[1] from Dudley's statistics of the Royal Naval School showing a somewhat similar epidemic prevalence of influenza, whilst there is a fair amount of tonsillitis, which was so prevalent at "X" (it must be noticed that tonsillitis has only been recorded from the summer term of 1916). There are several interesting differences: scarlet fever and diphtheria appear in epidemic form, but measles and chickenpox are almost entirely absent (nothing under 1 per cent. is shown) and German measles appears once, and then with an attack-rate of only 5·5 per cent. This comparative absence of measles, German measles, and chickenpox may perhaps be explained by the fact that the Greenwich boys come from the public elementary day schools, instead of from private boarding preparatory schools, and so probably have had most of these diseases before their entry at Greenwich, usually at the age of 12. The heading feverish cold, chill, etc., is apparently not recorded in the Greenwich statistics.

The next diagram[1] I show is a year diagram, constructed from Dr. Dukes' Rugby statistics from 1871 to 1904 ("Health in Schools," p. 467). It has many points of interest, which include the absence of influenza before 1890, and even after that year the comparatively small size of the attack rates for this disease. Compared with Greenwich, the almost biennial visitation of measles, the frequent appearance of German measles and the much greater prevalence of mumps, which all tend to offset the comparative absence of scarlet fever and the complete absence of diphtheria are noticeable. The celebrated "fourth disease" appears in history in 1900.

Returning to the diagram (fig. 2) of "X," large proportions of admissions are due to the following:—(1) Influenza (28 per cent.). (2) Feverish cold, chill, pyrexia and bronchial catarrh (15 per cent.). (3) Tonsillitis 20 per cent. of all admissions for the four years under review. The three added together and compared with Simey's[2]

[1] Diagrams not reproduced.
[2] *Brit. Med. Journ.*, 1922 (i), 134, and *Journ. Roy. San. Inst.*, January, 1927, 480.

published figures for "febricula" at Rugby (which term he uses to include all three), even after allowing for the absence of house sick-rooms at "X," show an increase, due in part to persistent infection with hæmolytic streptococcus, which has been endemic at "X," but partly due, I think, to the three factors already suggested.

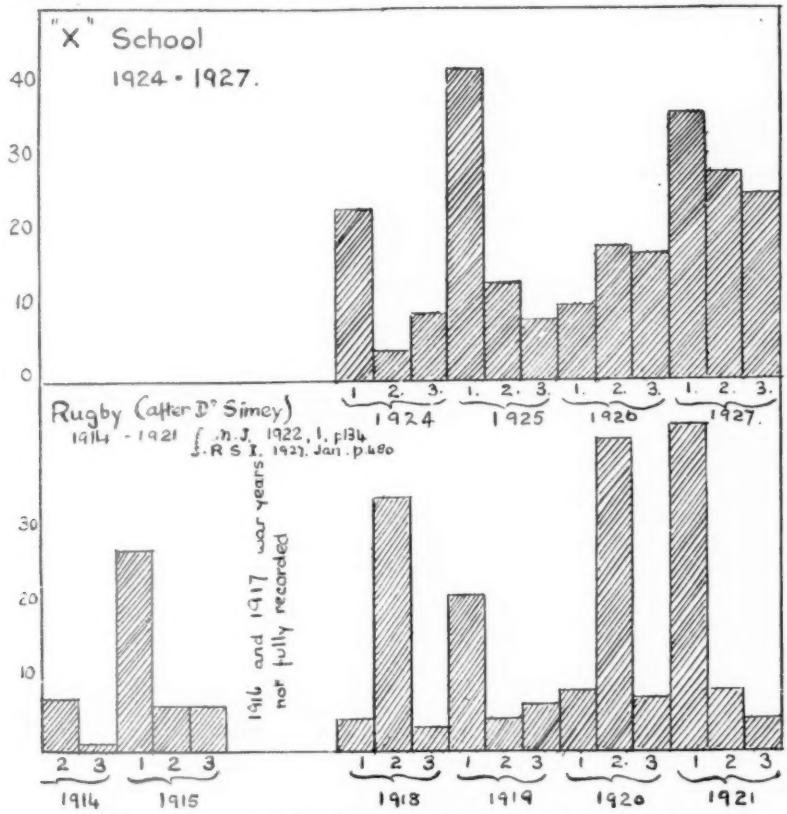

FIG. 3.—Diagram showing "febricula" attack rates per cent. per term.

Some apparently regard it as almost impracticable to distinguish between epidemics of influenza and those of febrile colds, but we should try, though difficulty often arises.

THE COMMON COLD.

Dr. Lempriere [1] states that colds and feverish catarrh account for 25 per cent. of the admissions to the sanatorium—excluding epidemics of influenza. This agrees with the figures of the causes of absence for six public elementary schools in Sheffield, where 30 per cent. of the attendances, lost on account of illness, were due to coughs, colds, bronchitis, etc. These figures covered a period of eight weeks in February and March, 1921, and showed that 80 per cent. of the absence was due to illness, confirming the opinion that in public elementary schools roughly 10 per cent.

[1] *Journ. Roy. San. Inst.*, January, 1927, 485.

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of school time is lost owing to illness. In Sheffield 10 per cent. of the sick absence was due to zymotic diseases, 6·7 per cent. to contagious diseases, and 30 per cent. to coughs, colds, bronchitis, etc., while 3·8 per cent. was due to sore throats. The period in question was not one of influenza epidemic in Sheffield.

Dr. D. K. Brundage[1] has recently published a valuable paper analysing the sickness records of the Edison Company of Boston for ten years, ending December, 1924, and showing the comparative importance of the common cold and the other respiratory diseases in producing disability in adults.

These duration figures correspond fairly with figures in English public schools, the usual time in sanatorium for influenza cases being about ten days (1925, 10·35 days; 1926, 11·78 days; 1927, 9·36 days). Appended is a table of comparison between the Edison experience and that at "X."

ANNUAL EXPERIENCE PER 100.

	Edison Company, Boston, ten years ending December, 1924, all absences of one day or over due to sickness	"X" School four years ending December, 1927, all admissions to Sanatorium
Due to all diseases	101	95
Proportion due to respiratory diseases ...	54 per cent. of all absences	68 per cent., or 88 per cent. if measles and German measles be included.
Days lost per absence for influenza ...	9·8	10. (This figure is based on three years' experience at "Y" School.

INFLUENZA.

We are often in doubt whether an epidemic of febrile catarrh is influenza or not, but, as a rule, when the real epidemic comes there is no doubt about it; the character of the epidemic is a better guide even than the clinical character of the case. The true influenza epidemic is of an explosive character, which was early remarked upon. Sir Thomas Watson wrote, in 1833: "On April 3 I saw the first two cases that I did see of the influenza; all London was smitten with it on that and the following day." Figure 4 illustrates this explosive character in a school epidemic reported on by Dr. Copeman.[2] A similar explosive epidemic may be witnessed in a day school. In 1890, for example, half the boys of Chesterfield Grammar School were attacked by influenza on the morning of Monday, January 27.

High attack-rates are very common, and in certain houses I have known the attack rate reach 95 per cent. The diagram gives some of these for various years.

There seems to be little or no correspondence between the height of the attack rate in an influenza epidemic and the prevalence of complications, such as pneumonia and otitis media. The pneumonia and otitis media are in fact secondary epidemics which may or may not occur. Thus, in 1890,[3] on the training ship "Shaftesbury" 52 per cent. of the boys were attacked with influenza in eight days, but no case of pneumonia occurred. The following April, 1891, there was another epidemic; only 25 per cent. were attacked, but there were five cases of pneumonia. Dr. Lempriere has said that true influenza is beyond the control of the school medical officer, but that this ought not to apply to that most persistent endemic condition, P.U.O.

We must now ask ourselves if there is any immunity to be obtained against influenza; does, for example, a previous attack, or the use of vaccine, imply immunity? In the great epidemics of 1918 and 1919 there seemed to be evidence that a previous attack did confer some immunity against one or more of the subsequent waves of infection. Thus, at King's College School, Cambridge,[4] where 100 per cent. of the boys were attacked by the epidemic in July, 1918, none were

1 *Public Health Reports*, Washington, 43, 11, March 16, 1928.
2 *Ministry of Health, Report No. 4*, Influenza, 411.
3 Dr. H. Franklin Parsons, *Second Report on Influenza*, L.G.B., 1893, 76.
4 *Ministry of Health Report No. 4*, Influenza, 400.

attacked in the epidemic of November, 1918, but 58 per cent. were again attacked in March, 1919. A somewhat similar sequence occurred at Rugby.[1] In the July epidemic of 1918, 37 per cent. were attacked, in the November epidemic of the same year only 3 per cent., whilst in the March, 1919, 23 per cent. were attacked, those who had had the disease in July, 1918, epidemic having practically the same attack-rate as the school as a whole. These, attack-rates only include admissions to the sanatorium. Dr. Simey,[2] in 1922

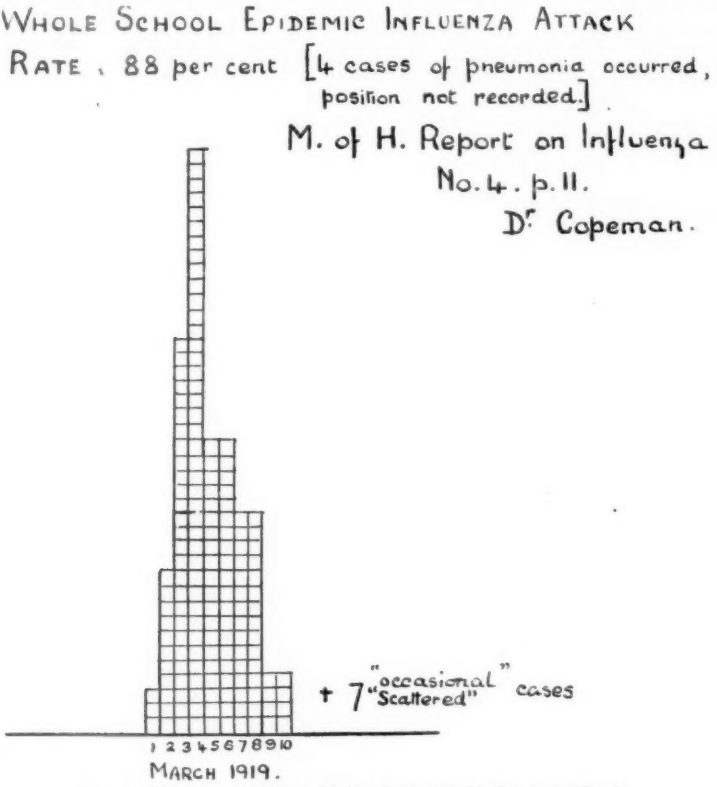
WHOLE SCHOOL EPIDEMIC INFLUENZA ATTACK
RATE . 88 per cent [4 cases of pneumonia occurred, position not recorded.]
M. of H. Report on Influenza
No. 4. p. 11.
Dr Copeman.
+ 7 "occasional" "Scattered" cases
1 2 3 4 5 6 7 8 9 10
MARCH 1919.

FIG. 4.—Diagram showing explosive character of influenza epidemic.

considered that partial and temporary immunity is conferred by an attack of febricula against influenza, and by influenza against febricula. I am interested to know if he still holds this opinion, for in recent years it has seemed to me that no immunity is conferred by attack, but rather the reverse. In several epidemics recently there has been a tendency to a second attack only three or four weeks after the first; nearly 50 per cent. of the boys in one house had definite second attacks in the same term in one epidemic which I investigated in February-March, 1925. Here is the experience of a preparatory school of fifty-eight boys. In the Christmas term of 1924, 52 per cent. of the boys were attacked with influenza. In the Christmas holidays and the following Lent term twenty of those thirty

1 *Brit. Med. Journ.*, 1922 (i), 134. 2 *Op. cit.*, 135.

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who had been attacked in the Christmas term were again attacked by influenza; whilst of the twenty-eight who had escaped in the Christmas term, 14 or 50 per cent. were attacked, so that the figures are against immunity being conferred by the first attack.

There is pretty general agreement that true epidemic influenza cannot be prevented by vaccines, but there is considerable difference of opinion as to whether the common or feverish cold, P.U.O., or febricula can be prevented, and whether the incidence of complications such as pneumonia and otitis media can be diminished.

As to the efficacy of vaccines in the prevention of influenza or febricula in public schools, there is some little published evidence of a somewhat conflicting character, Dr. Lempriere's[1] distinctly favourable observations at Haileybury and Dr. Attlee's,[2] at Eton, which are much less so. By the kindness of Dr. Graham Forbes, I have seen the summary of the evidence produced by a questionnaire sent out by the Medical Officers of Schools Association. In one or two epidemics with the assistance of my colleagues, Dr. Griffith and Dr. Scott, I have in two schools tried out special vaccines made from strains of organisms actually found at the schools in recent outbreaks. The evidence is conflicting and I think entirely inconclusive. One thing must, however, be remembered: the scales are weighted against vaccines by the fact that boys specially prone to influenza and colds are more likely to be vaccinated than boys who do not suffer from colds. In answering the questionnaire thirteen medical officers thought that vaccines did good and thirteen others considered them of little or no value. Strangely varying opinions were expressed. One medical officer, for example, says: "Cases of catarrh are fewer than in previous years. I believe the inoculations have done much good." Whereas another says: "No inoculations—only thirty-eight out of school, average, four: if the whole school had been inoculated the low incidence would have been attributed to this. I do not consider prophylactic procedure justified by the figures." One observer considers that "vaccines are useless in the presence of chronic nasal trouble"; whereas another observer states that he has "particularly noted good results from vaccines in adenoid cases." On the question of *the prevention of complications* in one epidemic, I had the opportunity of observing in the Lent term of 1927, a school of 450 boys in which there were 163 cases of influenza (an attack rate of 36 per cent.) and fifteen cases of pneumonia, five (i.e., all those which were typed) of which were due to Type I pneumococcus, whilst the attack-rate of the inoculated (32 per cent. of the total population had been inoculated with various stock vaccines) and of the uninoculated (68 per cent.) was almost exactly the same. None of the boys, however, who developed pneumonia had been inoculated. In November, 1927, 71 per cent. of the boys of this school were given a special vaccine[3] prepared in the Ministry's Pathological Laboratory, containing pneumococcus Types I and II, and although an outbreak of influenza occurred in the Lent term of 1928 with almost the same attack-rate as in 1927 (36 per cent.) no cases of pneumonia occurred. But the uninoculated 132 boys not only had a slightly lower attack-rate for influenza (23 per cent.) than the 319 inoculated boys, whose attack-rate was 30 per cent. (boys only inoculated once omitted), but they also like the uninoculated had no cases of pneumonia. In another school in the same term, where 90 per cent. of the total population had been vaccinated with a special vaccine of Pfeiffer's bacillus with pneumococcus Types I and II, prepared in the Ministry of Health Laboratory from strains similar to those

1 *Brit. Med. Journ.*, 1927, April 2; October 29.
2 *Brit. Med. Journ.*, 1927, October 15, 702.
3 This vaccine contained pneumococcus, Type I, 200 millions in 1 c.c., pneumococcus, Type II, 100 millions; *Streptococcus hæmolyticus* (Dochez) 60 millions. A form of tonsillitis due to infection with hæmolytic streptococcus (Dochez) had been common in the November term of 1927. The following term tonsillitis recurred in epidemic form, but Dochez was not identified, although several other strains of hæmolytic streptococcus were.

TABLE II.—INFLUENZA ATTACK-RATES UPON ALL BOYS, INOCULATED BOYS AND UNINOCULATED BOYS RESPECTIVELY.

School Term and Year	All Boys			Inoculated Boys			Uninoculated Boys			Vaccines used and when given
	Number	Per cent. inoculated	Number attacked / Per cent. attack-rate	Number	Number attacked	Per cent. attack-rate	Number	Number attacked	Per cent. attack-rate	
Eton: Lent, 1927 [After Attlee]	1,019	30	443 / 44	302	149	49	717	294	41	Various "stock" mostly in Xmas holidays
Haileybury: Lent, 1927. [After Lempriere]	530	40	101 / 19	215	34	16	315	67	21	Parke Davis A.C. 95. "Public School" 120
"X" Lent, 1927	447	32	163 / 36	144	54	38	303	109	36	Various in Xmas holidays
"X" Lent, 1928	463	71	133 / 30	331	102	31	132	31	24	Special M. of H. in Nov., 1927
"Y" Lent, 1926	38	56	44 / 12	212	..	13	168	17	10	Various in Xmas holidays
"Y" Lent, 1927	383	91	72 / 19	347	62	18	36	10	28	Special M. of H. Vaccine given in November term, 1927
Totals	3,222		956 / 30	1,551	42	28	1,671	528	32	

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found in the previous year's epidemic, and administered in the previous November by the school medical officers, there was an almost complete absence of complications, but that had been true also of two previous epidemics, in one of which stock vaccine had been used for 56 per cent. of the total population, and also in much more severe previous epidemics of which we had no information as to the vaccinal condition. My own feeling with regard to vaccine against influenza and febricula is that the question is still entirely open, but if it is determined to recommend prophylactic inoculation against influenza, colds and their sequelæ, it is important that the course of vaccines should be given before the epidemic prevalence begins, preferably in November. This means that the injections should be given at school, and this requires a good deal of rearrangement of time tables, but my experience is that if the injections be left to the Christmas holidays they are as a rule deferred to the period of epidemic prevalence which usually begins about the 14th to 21st January.

Bacteriological Findings.

Drs. Griffith and Scott have examined many nasopharyngeal and tonsillar swabs taken by me in school epidemics. At one school, "Y," epidemics in four years were studied, the findings being set out in the following table:—

TABLE III.—"Y" SCHOOL BACTERIOLOGICAL FINDINGS.

Year	Number of swabs	Pfeiffer's bacillus		Pneumococcus						Other
		Present in	Carrier rate	Present in	Carrier rate	Types				
						I	II	III	IV	
1920	19	6, all serologically identical with 1 Lister strain	32	11	58	not determined				Meningococcus in 1
1925	43	20	46	27	63	1†	0	5	21	Meningococcus in 3 (8 per cent.)
1926	52	25	48	24	46	0	0	2	22	Meningococcus absent: hæmolytic strep. in 1
1927	43	32 (27 very profuse) 72 per cent. no indol formation	73	28	65	1†	0	8	19*	Hæmolytic para influenza bacilli in 2, hæmolytic streptococcus absent, meningococcus absent

* 4 Pn 85, 1 π B, 1 Pn 10, 1 Pn 160 } Agglutinated with monovalent sera prepared from strains isolated from cases of pneumonia.
12 not serologically identified.
† The two Type I pneumococcus were found in carriers who had not had pneumonia.

Previous to the 1927 investigation a vaccine (Pfeiffer's bacillus with Pneumococcus I and II) prepared at the request of the school authorities in the Ministry's Pathological Laboratory had been given in November, 1926, i.e., before the epidemic season began, to 90 per cent. of the boys.

It is interesting to note that at "X" in the Lent term, 1927, when some fifteen cases of pneumonia occurred, in every case (5) in which the pneumococcus was typed, the type found was Type I. Type I is sometimes termed the "pneumococcus of youth."

PROPHYLAXIS, OTHER THAN VACCINES, AGAINST INFLUENZA.

I am convinced that a more intensive prophylaxis against influenza and epidemic catarrh at the beginning of each Lent term (i.e., during January and February) would well repay school authorities. Apart from the experience of most schools and all

other experience, the Edison Company's Charts, by Dr. D. K. Brundage,[1] show admirably the periods of maximum incidence in January and February when it seems that our chief efforts should be made. I recommend a special letter to parents and a special form of certificate for the Lent term. I have not seen any health certificate that mentions influenza or feverish cold, and I suggest that the Lent term certificate might be printed on red paper to distinguish it, and should include a statement that the boy had not had a severe or feverish cold for a week prior to the day of return. I have seen three striking examples of the mischief that boys can do by returning to school prematurely after influenza. Here is one: One of two brothers contracted mild influenza at home in London on January 13, 1925; the boy travelled by the ordinary school train on January 16 in a compartment with seven other boys and had dinner in the dining car. The seven boys in his compartment (four belonging to his own house) and the boy who sat next to him in the dining car fell ill with influenza on the 18th or 19th. His brother, who may have been infected at home, became ill on January 17. The boys of the house to which this boy belonged almost all suffered severely from influenza, the attack-rate being 95 per cent., and many of the inmates had two attacks. The attack-rate in this house was by far the highest of any house in the school.

Temperature Taking.

I see no reason why the temperature of all boys should not be taken for the first three weeks of the Lent term, as is done in so many preparatory and girls' schools. Girls are certainly no less susceptible than boys, as is proved by evidence from co-education schools and from orphanages, etc., where boys and girls are under the same roof. Most of the evidence, including the Edison Company's experience before quoted, is, I think, that females are slightly more susceptible, yet the girls' schools appear to escape more lightly than the boys' do. This is, no doubt, largely because girls usually sleep in cubicles, and the girls' schools are, as a rule, of more modern design than boys' schools, and partly because girls are more under control than boys, but it is also, I believe, very largely due to the practice of taking every girl's temperature every night and morning for the first three weeks of term and to the immediate isolation of any girl with a slight pyrexia. Why a scientific procedure like taking a temperature should be regarded as "coddling" or "fussing" I cannot say.

Isolation of Slight Pyrexia.

The immediate isolation of any severe catarrh, or slight pyrexia, occurring within the first three weeks of term is of great importance.

Early School.

I am strongly of opinion that there should be no work before breakfast, during January, February and probably March, though I should have no objection to a short sprint in the open air.

Not only are boys' resistances I believe at a lower level before breakfast (admittedly a point difficult of demonstration) but the heating of the classrooms is usually at an even lower ebb, a point which can often be easily shown. I have seen classrooms which started the day at 36° F.

Nasal Douching and Sprays.

Much previous experience with the meningococcus has rendered me sceptical as to whether good results follow the use of nasal douches or sprays. I have, however, met one schoolhouse in which their use appeared to be successful, and Dr. O'Brien[2]

1 *Public Health Reports, Washington*, March, 1928.

2 *Journ. Roy. San. Inst.*, January, 1927, 485.

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has expressed "a considerable amount of faith" in the power of an antiseptic oily spray to give a reasonable amount of protection. Gargling I regard as a harmless procedure in catarrhal conditions, and possibly useful in tonsillitis, but I should not recommend any form of nasal douche or spray as a routine measure for healthy boys; "nasal drill" is less objectionable, but I am not enamoured of it.

Baths.

It seems probable that a proper skin reaction is one of our greatest defensive mechanisms against chill, and I strongly urge, with this end in view, that all hot baths or hot showers should be followed by cold showers. The Scotch douche (alternating hot and cold showers) might be well worth a trial as a prophylactic measure, especially for well-known susceptibles, and artificial sunlight might be well worth a trial for such boys as are known for their liability to colds.

Diet.

It appears to me important that in the Lent term fresh fruit, particularly oranges, or fresh vegetables such as salads, watercress, etc., should be taken daily.

Prevention of Infection.

This is my main theme. Where does infection usually take place? There can be no doubt that by far the majority of infections take place in the dormitories. I have investigated the possibility of infection in classrooms, dining halls, chapels, etc. In comparing incidence in badly ventilated classrooms with that in better classrooms, I found that the attack-rate in the poor classrooms was 38 per cent., in the good 32 per cent., but as the younger boys were, as a rule, in the poor classrooms, and the older classes in the better ones, the age-distribution may have accounted for this difference, although it is not always the younger ages which suffer most heavily in influenza epidemics in schools.

In an epidemic which I investigated some years ago, in a great public school, most of the infection apparently occurred in common rooms which were used alike for meals, for preparation of work, and for the accommodation of convalescents from influenza; the windows of these rooms could be opened for a few inches—conditions which not unnaturally caused disastrous results although the sleeping accommodation was admirable and on the cubicle system.

Infection in Sleeping Quarters.

But I am convinced that the main body of infection is conveyed in sleeping quarters. Where else do the infecting boy and the recipient of infection maintain close proximity for as long as the ten hours they spend in the dormitory? Again, the movement of air is less than during the day owing to the cessation of movement and the shut doors, and, unless the ventilation is really efficient, pockets of stagnant air form (particularly in corners).

Many boys sleep open-mouthed, the nasal filter is short circuited, and the recipient receives the droplets of secretion containing the infection, particularly when sprayed by cough or sneeze, through the open mouth. The way to minimize this infection is of course to keep the beds far apart with efficient cross ventilation.

One of the things which have struck me in public and private boys' schools is the comparative crowding of beds which I have frequently encountered. The Royal Commission on Barracks which reported in 1861 insisted that there should be a distance of 3 ft. between the edges of beds; 60 square ft. floor space and 600 cubic ft. per bed.

The Board of Education in 1914 in issuing their Building Regulations[1] for Secondary Schools, laid down the principle that there should be a space of at least

1 "Cd. 7535." Stationery Office.

3 ft. between beds, a floor area of not less than 65 square ft. and an air space of 700 cubic ft. for each occupant; that ventilation must be adequate and that it is very desirable that a through current of air should be provided by arranging the windows on opposite sides of the room. Dr. Dukes[1] lays down the standard of 800 cubic ft. Nevertheless in my experience it is quite an exception to find 3 ft. space between beds. I have found, for example, six beds in 27 ft. of wall space, and this in dormitories which were not cross ventilated, and I have found dormitories ventilating into each other and even into classrooms; dormitories with fifteen beds only 1 ft. 4 ins. between beds with 52 square ft. of floor space. In these crowded rooms, direct bacteriological evidence of cross infection with hæmolytic streptococcus was found, reminding one of the cross infection with meningococcus which one used to find in an overcrowded barrack room.[2]

In the bed plan [shown] of a certain public school dormitory showing the grouping of cases of influenza (attack-rate 50 per cent.), it may be coincidence, but the beds are less than 2 ft. 6 in. apart, ventilation is "end" and not "cross," and there is much "dead space." In a dormitory from another school [plan shown], beds are 22 ins. to 18 ins. apart, the ventilation is poor, total floor space only 48 square ft. per bed, the effective floor space less. Eleven of the fifteen boys were swabbed, and five of the eleven were carrying hæmolytic streptococcus, a carrier-rate (non-contact) of 45 per cent.

Tonsillitis.

I have investigated persistent epidemics of sore throat in three schools. In one the condition seemed due to non-hæmolytic streptococci, and there was a possible connexion with some sanitary defects; the infection at least cleared up immediately these conditions were rectified.

In the other two the infection was due to hæmolytic streptococci. In one of these schools it was a long series of "dropping" cases of scarlet fever that caused attention to be drawn to the infection, in the other the tonsillitis was the feature of the cases which attracted attention and there was only one sporadic case of scarlet fever, although many of the patients with tonsillitis carried strains of hæmolytic streptococci which were serologically identified by Drs. Griffith and Scott with strains obtained from cases of scarlet fever. In one term nearly all the cases examined carried a hæmolytic streptococcus identical with a strain isolated in New York by Dochez; this strain is associated with scarlet fever but not, as far as is known, with it in epidemic form. It seemed possible at the school where the dropping cases of scarlet fever occurred that we were seeing a phenomenon ("the warning rise") rather similar to that seen in cerebro-spinal fever[3] (and possibly in diphtheria), i.e., that when the carrier-rate (i.e., the carrier epidemic) reaches a certain height (i.e., 20 to 30 per cent.) clinical cases may occur.

In this school thorough "spacing out" of beds was carried out, together with improvement of the ventilation of the dormitories, and since then, no more cases have occurred, six months having now elapsed (fig. 5).

The observations were, however, too fragmentary for this to be anything but a surmise.

This infection with hæmolytic streptococci was in one or two instances I think definitely due to unsatisfactory conditions (overcrowding and poor ventilation) in sleeping quarters.

In connexion with tonsillitis the question of milk must be raised. I have only dealt with three samples of public school milk and all were definitely dirty and bacteriologically bad. Other evidence seemed to show that the milk was not

1 "Health at School," 1905 (Rivingtons) p. 140.
2 Glover, *Journ. of Hygiene*, xvii, 1918, October 31, 378.
3 Glover, *M. R. C. Special Report* No. 50, p. 140.

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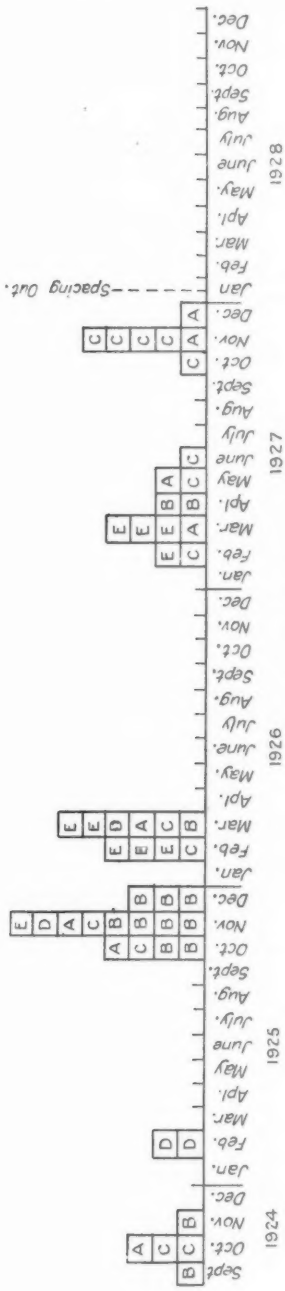
Spacing out.
Sept. Oct. Nov. Dec. Jan. Feb. Mar. Apl. May June July Aug. Sept. Oct. Nov. Dec. Jan. Feb. Mar. Apl. May June July Aug. Sept. Oct. Nov. Dec. Jan. Feb. Mar. Apl. May June July Aug. Sept. Oct. Nov. Dec.
1924
1925
1926
1927
1928
Monthly Columns.

Dropping" cases of scarlet fever: diagram showing "House" distribution and cessation after "spacing out" of dormitories
8 cases from "A" House; 14 cases from "B" House; 14 cases from "C" House; 10 cases from "D" House.

FIG. 5.

responsible for the tonsillitis, but apart from this, I should not feel happy as a school medical officer unless either pasteurized or Grade A milk were in use. Pasteurization is best done on delivery at the school premises, as at Dartmouth.

I trust that medical officers of schools will forgive me if I suggest that there are great opportunities for interesting epidemiological study still unexplored in public schools. I suggest as examples:—

(*a*) The epidemiology and bacteriology of otitis media in relation to influenza and febricula.

(*b*) Relative sickness-rates of boys' and girls' schools and of day and boarding schools.

(*c*) Relative incidence of droplet infections upon children whose tonsils have been enucleated and whose adenoids have been removed, compared with children who have not been operated upon.

The adoption or recommendation of some uniform system of medical records by the Association of School Medical Officers would be of great service. I hope that Association will renew its activities, which have been so helpful to me in preparing this paper, and keep before its members their paramount duty of devoting as much time and thought to the preservation of health in schools as they do to the care and treatment of the sick pupils. I urge, too, that the modern boy or girl is, and should be treated as, an intelligent being, and encouraged to take an interest and pride in keeping fit. This can be attained without fussing or coddling; public opinion nowhere has greater influence than in schools, and it should be formed to regard the training of the body against infection as being as manly an aim as its training to achieve athletic success or to endure fatigue.

In the following summary of my points I advance them not as "conclusions," or as proved by my observations, but merely for convenience:—

(1) "Droplet" infections are responsible for at least 80 per cent. of all sickness in our public schools. There is a well marked "danger period," i.e., the first two months of the Lent term.

(2) It is probable that no immunity to influenza or febricula is conferred by previous attack, and evidence of the protective value of vaccines is inconclusive. There is, however, slight evidence that some protection may be afforded against complications, particularly pneumonia; the subject deserves more systematic study.

Vaccines, if given, should be administered well before the beginning of the danger period, probably best in November.

(3) Intensive prophylaxis during the first half of the Lent term would probably amply repay the trouble. It should include—

(*a*) Special efforts to prevent boys returning to school after the Christmas holidays infected with influenza or febricula.

(*b*) Temperature-taking for three weeks.

(*c*) Immediate isolation of all pyrexias and catarrhs.

(*d*) No work before breakfast for at least the first six weeks of the term.

(*e*) All hot baths and showers taken during the day or after games to be followed by cold showers.

(*f*) Prevention of chill in watching matches, sports, etc.

(*g*) Increased provision for drying clothes, uniforms and boots.

(4) Infection mainly takes place in sleeping quarters, and proper spacing out of beds and thorough "cross" ventilation in dormitories are of paramount importance. No school authority has done its duty to its pupils unless it has provided dormitory accommodation sufficient to allow of at least 3 ft. clear space between the edges of beds, and thorough and through ventilation. Until these essential wants are met, the provision of properly sited, amply spaced and "cross" ventilated dormitories should take precedence of all other building requirements.

(5) Milk should be pasteurized.

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Discussion.—Dr. S. MONCKTON COPEMAN (President) said that Dr. Glover's paper would undoubtedly give rise to considerable discussion. Not having had much personal experience of school work, he had been surprised to learn that no fewer than 80 per cent. of all cases of illness were due to "droplet" infections rather than to ordinarily recognized infectious diseases. It was somewhat difficult to follow Dr. Glover's explanations of the comparatively small incidence of the usual infectious diseases of childhood—such as diphtheria, scarlet fever and measles—in preparatory and public schools, and in public elementary schools and training ships respectively. For whereas in the case of the public schools Dr. Glover regarded the low level of infection as due to the fact that care was taken in the home surroundings to avoid exposure to infection; in the case of public elementary schools and training ships he ascribed it to the probability of immunity having been established by continual minimal exposures to infection in home surroundings. Another point which appeared to require further elucidation was the relative importance of dormitories and class-rooms in the spread of infection. He (Dr. Copeman) was of opinion that in certain conditions, such as overcrowding and bad ventilation, the possibility of infection in class-rooms might be by no means negligible. He had, in the course of an official inspection some years ago, obtained conclusive evidence of the extension of infection of diphtheria from the original case in one of the class-rooms of a public elementary school.

Surgeon Commander S. F. DUDLEY said that increase in droplet infection since the war was not shown in naval statistics. Even if it was possible it was doubtful if it would be wise to prevent all droplet infection. Schools taught the individual how to combat bacterial attacks in after life. In many infections resistance to attack was gained without symptoms, by the inhalation of subinfective doses of infective material. Halliday had shown this process of autovaccination to occur even in measles. The ideal would be to produce this immunity by the correct amount of contact. In our present state of knowledge this was hopeless, and where sanitation and nutrition were average there was little to show that isolation, artificial vaccines, special diets, douches or other "placebos," had any appreciable effect on the incidence of the minor droplet infections. He would therefore support Dr. Glover's appeal that we should concentrate on the factor that undoubtedly had a beneficial effect on lowering the morbidity of many infections. This factor was increase of space, especially in sleeping quarters. One example would suffice to illustrate the importance of dormitories. In a school where boarders and day boys associated closely during the day the morbidity during three years had been 30 per cent. among the former boys for diphtheria and scarlatina (combined), while not a single day boy had contracted either of these infections. Unfortunately, certain types of influenza and the minor non-specific droplet infections seemed to spread almost as easily in a crowded classroom as in a dormitory. He was, however, convinced that the most hopeful method of reducing all infections in schools was to concentrate on increasing the wall space of the sleeping quarters and diminishing the number of inhabitants per room. Was there any reason, except old-fashioned prejudice, why all public school boys should not sleep in open-air shelters?

Dr. G. E. FRIEND said that fifteen years ago, when he began work at Christ's Hospital, he had been alarmed by the numbers of boys taken sick, but he had since found that his own case-incidence corresponded very fairly with that of the few schools from which figures were available; the numbers given by Dr. Glover supported this. In his (Dr. Friend's) experience there had been no increase in the total number of these cases from 1903 to 1927. On the contrary, there had been a small decrease. His figures were:—

	Total medical in-patients, including all infection	P.U.O, tonsillitis, etc.	Percentage
1903 to 1907 ...	6170 ...	? ...	?
1913 to 1917 ...	6132 ...	1533 ...	25
1918 to 1922 ...	6246 ...	1372 ...	22
1923 to 1927 ...	5255 ...	1296 ...	24

He found by the use of spot maps of dormitories, class-rooms and house day-rooms, that infection more usually occurred in the latter rooms and *not* in the dormitories. In measles and influenza when infection was explosive the dormitories seemed equally responsible with the class- and day-rooms, but not in other infections. He attributed this to the size of the rooms and good bed-spacing. There was four times the space per boy in the dormitories as in the class- and day-rooms, and the dormitories were built like hospital wards with outside sanitation and both cross and end-to-end ventilation directly from the outer air.

He had not tried mass vaccines for P.U.O., but had done so for influenza in October, 1918 and February, 1920, using a vaccine of various strains of Pfeiffer only. On the first occasion with 73 per cent. of the boys protected no influenza appeared, and the same thing happened in 1920 when 92 per cent. were protected, though influenza was prevalent around and several cases occurred among the staff and servants, few of whom were protected. The results of the first inoculation were reported in the *Lancet*, January, 1919.

He did not consider mass inoculation practicable in a large school. To be effective it must be done during term, and the necessary disorganization of games and routine, and the difficulty of getting so large a number of temperatures taken efficiently, prohibited its employment except as an emergency measure.

He agreed with Dr. Glover in regard to the other means of prophylaxis. Intensive prophylaxis had been carried out at Christ's Hospital for some years on all the lines mentioned except the taking of temperatures, and though the mass results so far were not marked there was some improvement especially in the number of cases of P.U.O.; he hoped for further improvement though agreeing with Commander Dudley that a very great reduction might not be wise even if it were possible.

The measures he personally employed were: (1) Lectures on personal hygiene; (2) rules of health posted in all dormitories; (3) nasal drill in dormitories night and morning; (4) supervision of changing after games, etc. (this varies much in different houses); (5) follow-up dental inspection; (6) close inspection of milk supply (the school has its own herd); (7) close supervision of bathers as well as of swimming bath, with the exclusion till clear of all cases of septic trouble, grazes, and especially otitis and otorrhœa. The provision of proper bathing caps for all old ear cases; (8) close supervision of dietary.

Professor M. GREENWOOD said he agreed with Surgeon Commander Dudley that so long as a herd continued to receive healthy non-immunes, it was not likely that any regulations, however good in themselves, would eliminate minor epidemic diseases.

With regard to the immunizing effect of a previous attack of influenza in school populations, a good deal of information would be found in the sixth chapter of the Ministry of Health's Report on the Pandemic of 1918-19. The experiences of different schools were so widely discrepant that it seemed necessary to believe that different strains of organism differed widely in their powers of stimulating the production of antibodies. Thus, at Eton, only 7·4 per cent. of the boys attacked in the summer were again attacked in the autumn of 1918, while 47·8 per cent. of the boys who had been attacked in the summer were attacked in the autumn. The corresponding figures for Harrow were 32 per cent. and 76·1 per cent., and for Clifton 13·6 per cent. and 34·3 per cent.; on the other hand, at Haileybury the attack-rates were practically equal in the two classes—22·8 per cent. and 21·8 per cent. The practical conclusion seemed to be that only by a lucky accident could we derive much advantage from inoculation. On the whole, post-pandemic experience of artificial immunization against respiratory infections had not been encouraging. Professor Topley's Manchester experiment was very instructive in this connexion.

Captain W. DALRYMPLE-CHAMPNEYS said that the illustrations which he proposed to give had been mostly obtained during an investigation, the results of which had not as yet been published, lately carried out by himself at certain public schools.

(1) The first factor which he would emphasize in the spread of naso-pharyngeal epidemics in these schools was one which had already been dealt with by Dr. Glover, namely, overcrowding in dormitories. This was rather common in the schools he had visited. The following were examples of somewhat bad overcrowding:—

SPACE PER BED IN PUBLIC SCHOOL DORMITORIES.

School	Wall space	Distance between beds	Floor space	Effective air space (allowing 10 ft. in height)
A ...	5 ft. ...	2 ft. 6 in. ...	45 square feet ...	450 cubic feet
B (1) ...	5 ft. ...	2 ft. 6 in. ...	75 ,, ...	750 ,,
(2) ...	5 ft. ...	2 ft. 6 in. ...	80 ,, ...	800 ,,

Probably the most important dimension, from the point of view of epidemics, was wall space, but it was very frequently neglected, even when the floor space and air space per bed were fairly satisfactory.

The use of cubicles was effective in preventing the spread of infection, but only if the partitions were of sufficient height, and if cross-ventilation was secured by a window in every cubicle. He knew of two schools in which the cubicle partitions were only about

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3 ft. 6 in. in height. This was worse than useless, as such partitions merely prevented free circulation of air without preventing the spread of droplet infections.

(2) Secondly, overcrowding in the school sanatorium had an important bearing upon the incidence of secondary infections, such as pneumonia and otitis media. This defect also was common at the schools he had visited. The Voluntary Hospitals Commission had laid down the following standards for the main wards (not infection wards) of general hospitals:—

Wall space, 8 ft.; floor space, 96 sq. ft.; air space, 1,056 c. ft.

Judged by this standard the following examples from public school sanatorium wards showed severe crowding:—

Space per Bed in Public School Sanatorium Wards.

	Wall space	Floor space	Air space (allowing 10 ft. in height)
1 ...	6 feet ...	66 square feet ...	660 cubic feet
2 ...	5 ,, ...	51 ,, ...	510 ,,
3 ...	5 ,, ...	62·4 ,, ...	624 ,,

After careful consideration he suggested the following minimum standards for space per bed in the large (i.e., not observation) wards of school sanatoria: Wall space, 8 ft.; distance between beds, 5 ft. 6 in.; floor space, 100 sq. ft.; air space (allowing 10 ft. in height), 1,000 c. ft.

He suggested that for cases of infectious or respiratory disease the space per bed should be largely increased or even doubled.

(3) His third point was the waste of space due to bad planning in dormitories or wards, e.g., by unnecessary doors, or windows, or by their faulty disposition. This resulted in crowding beds into groups, which facilitated the spread of droplet infection.

(4) Lastly, he would draw attention to the neglect to space out the beds during epidemics. This practice had been observed by him not only at the public schools, but at all types of institutions for children. In many institutions in order to save labour some wards or dormitories were kept empty, whilst others were overfilled. He had recently observed a striking example of this in a Guardians' Home for Girls. This home was admirably kept and the children well cared for, but at the time of his visit, though nearly half the children had been removed to hospital suffering from diphtheria, the remainder were crowded into some of the dormitories, whilst other dormitories were empty. This home had nominal accommodation for forty children, but at the time there were only twenty-nine on the roll, eleven of whom were in hospital with diphtheria. The average space per bed in the occupied dormitories was as follows:—

Wall space, 4 ft. 6 in.; floor space, 35-40 sq. ft.; air space, 315-400 c. ft.

Dr. J. Tertius Clarke asked if Dr. Glover could offer any explanation of the fact that, though throat infections were almost the commonest cause of invalidity in the public schools, and though it was claimed that infected throats were the portal of entry of the virus of rheumatic fever, yet that disease was almost unknown in these schools.

Dr. Lewis G. Glover suggested that the Ministry of Health should organize, through the school doctors, an investigation into the use of vaccines as a prophylactic for colds. He would emphasize the following points: (1) That there were several different formulæ on the market; (2) That the doses of the vaccine varied considerably; (3) That the interval between the doses prescribed was also a very variable quantity.

Many of the failures seemed to be due to the vaccine not being administered early enough; one or two doses should be given in the summer, to protect against autumn colds.

Dr. Glover (in reply) said he was greatly obliged to Surgeon Commander Dudley for his powerful support and the convincing evidence of the day boy's immunity at Greenwich, a striking confirmation of the theory that infection usually took place in dormitories. The admirable charts which Dr. Friend had distributed of the dormitories at Christ's Hospital, with their ample spacing of beds, their 1,000 cubic feet of space per bed, and their perfect cross ventilation, fully explained why, at this school, infection took place mostly in the overcrowded day rooms rather than in such almost ideal dormitories.

He was quite unable to give the answer to the question propounded by Dr. J. Tertius Clarke. The absence of rheumatic fever in public schools, despite the prevalence of tonsillitis, was a remarkable phenomenon, and if an explanation could be found, it would add greatly to our understanding of the ætiology of acute rheumatism. Shaw had pointed out that the great reduction of rheumatic fever incidence in the Navy had not been accompanied by any reduction in the tonsillitis attack-rate.

War Section.

President—Surgeon Vice-Admiral A. GASKELL, C.B., O.B.E.

[*March* 12, 1928.

The Pathology and Treatment of Traumatic (Wound) Shock.

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ABSTRACT.—I.—Shock may be defined as "The clinical condition which follows an injury producing depressed vitality, associated with lowered blood-pressure, deficient circulating fluid, diminished intracellular oxygenation and reduced body temperature." Such a condition results from the presence of one or more of the following four factors, acting either singly or in combination: (1) Pain, (2) hæmorrhage, (3) cold, (4) toxæmia, either of bacterial, tissue, or other origin.

II.—Secondary traumatic shock is the common type and only becomes established slowly.

III.—Prevention is successful in many cases, even under front line conditions and in war surgery.

IV.—Treatment of an established case: (1) Application of warmth and mental rest. (2) Relief of pain, (3) restoration of deficient circulation, giving fluids by mouth, rectally, or by 10 per cent. glucose saline solution intravenously (1 litre in two hours). (4) Increase of deficient intracellular oxygenation by insulin hypodermically (5 units at beginning and end of glucose injection). (5) If operation is needed, by choosing a local or gas and oxygen anæsthesia.

CONTENTS.—(1) Introduction—(2) Conception of Shock before 1917—(3) Work of Shock Committee (Medical Research Committee)—(4) Summary of Front Line Observations—(5) Pathology of Circulation in Shock—(6) Toxæmic factor in Shock—(7) The Central Nervous System—(8) Metabolism in Shock—(9) Present Conception of the Pathology of Shock—(10) Treatment—(11) Bibliography.

I.—*Introduction.*—Interest in the subject of traumatic shock has recently revived. At a meeting of the Section of Surgery, held early in this year, a criticism of current views was made by Mr. Zachary Cope and discussed by several surgeons.

It was agreed that although our knowledge of the subject is now reasonably complete, the acceptance of such views is still far from universal and the teaching in current text-books is confusing and even misleading. Authorities have not agreed on a precise definition of "shock" and find difficulty in clearly differentiating between "shock" and "collapse." At the sixth meeting of the International Society of Surgery (London, 1923), Professor John Fraser, of Edinburgh, read a valuable paper on "Operation Shock" [1].

Fraser's definition is not quite comprehensive, but is one of the best hitherto put forward.

"Shock" may be defined as a state of depression of all the vital functions of the body, the state being primarily induced by the infliction of injury on the body tissues, and being characterized by a progressive fall of the blood-pressure.

Cope's definition is a little fuller, but omits to mention the blood-pressure.

The term "shock" signifies a condition following the application of harmful stimuli, or the depletion of the body fluids, in which there is a serious and clinically demonstrable depression of the vital processes of the body, particularly the circulation and metabolism.

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It is difficult in a short, polished phrase to give a complete verbal description of what is meant by "shock."

Cannon says [2]—

> It seems to me that in such a complex as "Shock," definition is not a prime requisite. The important matter is to obtain a careful description of the observed facts.

For practical purposes, however, it is useful to epitomize the pathology of the condition in a few words. I would suggest the following:—

> By "shock" is meant that clinical condition produced by an injury which induces depressed vitality, associated with lowered blood-pressure, deficient circulating fluid, diminished intracellular oxygenation and reduced body temperature. Such a condition results from the presence of one or more of the following four factors, acting either singly or in combination: (1) Pain. (2) Hæmorrhage. (3) Cold. (4) Toxæmia, either of bacterial, tissue (protein), or other origin.

II.—*Conception of Shock before 1917.*—The early view of the pathology of shock was that it was of nervous origin.

Malcolm, Lockhart-Mummery, Crile and others believed that shock was a nervous phenomenon, associated with peripheral vaso-constriction, splanchnic dilatation, or exhaustion of the cerebral cells. A deficiency of circulatory fluid was recognized, but further details were not known. A large number of theories existed, but actual observed clinical facts were few, and definite pathological details almost unknown.

III.—*The Shock Committee.*—An immense stimulus to those interested in shock was provided by the publication of the Medical Research Committee's "Memorandum upon Surgical Shock and some Allied Conditions," in February, 1917. The work of Dale and Laidlaw on histamine shock, and to a lesser extent, perhaps, that of Bainbridge and Trevan on adrenalin injections, opened up a fresh field of thought. The Shock Committee coördinated research, infused fresh enthusiasm in the minds of the workers concerned, and at the end of the year (1917) published many valuable original observations on the condition.

In France a group-research was organized in the First Army area. Cannon and John Fraser worked in the laboratory and surgical wards of a C.C.S. in Bethune. My part of the work in the front line and forward areas was to make observations on the early condition of casualties which were subsequently evacuated to this C.C.S. In this way the complete histories of many cases of shock were obtained, observations being made before wounding, at the time of wounding, and at periodical intervals during the stage of evacuation.

IV.—*Summary of Front Line Observations.*—From these clinical studies, the factors which predominate in the pathogenesis of wound shock appear to be: (1) Pre-wound factors of fatigue, exposure, lack of water, loss of bodily fluids and presence of excitement. (2) Post-wound factors of pain, hæmorrhage, cold, and adsorption of bacterial or tissue toxins.

V.—*Pathology of the Circulation in Shock.*—(*a*) The quantity of fluid circulating in the vascular system is diminished. (*b*) The concentration of blood is increased and capillary stasis is present.

(*a*) In the spring of 1918 N. M. Keith [6] and O. H. Robertson [7] measured the blood-volume directly by the vital red method in thirty cases of shock. In cases with distinct symptoms of shock the blood-volume ranged from 51 to 85 per cent. of the normal, while there was a corresponding reduction of the plasma. In one striking fatal case this loss of blood-volume occurred when there had been no hæmorrhage whatever.

From these studies Keith makes a clinical division of shock cases into three groups. (1) *Compensated Cases*, in which beyond pallor, weakness and a slightly quickened pulse, the general condition is good, the blood-pressure remains above 100 and the blood-volume is never reduced below 80 per cent. (2) *Partially Compensated Cases* in which the general condition is not good. There is usually a history of a smart hæmorrhage. The patient is pale, restless and thirsty, and readily vomits, he is cold, and has a rapid pulse and a blood-pressure usually 70 to 80 mm. The blood-volume ranges between 65 to 75 per cent. (3) *Uncompensated Cases*, in which the condition is extremely serious. The symptoms are even more pronounced than in (2) and the pressure has fallen below 60. On auscultation the heart-rate is 120 to 160. The blood-volume is below 65 per cent. of the normal. In normal men the loss of even as much as 800 c.c. of blood is followed by a quick return to the original volume. In shock this compensation fails to occur. This may be due to an anhydræmic condition of the tissues produced by diminished intake of fluids owing to stress of Service conditions and extra loss of fluid, violent bodily exertion, and sweating from various causes.

(*b*) Cannon and others found abundant evidence of concentration of blood in the capillaries, making, by blood-counts, observations on the hæmoglobin percentage and hæmatocrit readings. Even after hæmorrhage, if separate blood-counts of blood taken from a vein and from capillaries be compared, a discrepancy is found. In both counts the number of corpuscles is diminished, but more so in the veins.

This capillary concentration is regarded as being due to increased permeability of the capillary endothelium. Its exact mechanism, however, is not yet clear. As a result of this concentration the viscosity of the blood is increased, the capillary stasis still further encouraged, and a vicious circle is established.

Leonard Hill and McQueen [8] have studied the capillary circulation and especially stasis. They state that the primary cause of this condition is the fall of blood-pressure below 80, and the fact that the fall is not temporary, as in fainting. As the result of this stasis, de-oxygenation of the capillary areas follows. The osmotic pressure of the tissues rises, abstracting fluid from the capillaries. The capillary wall suffers and becomes more permeable and so the viscosity of the concentrated blood progressively increases.

Results of this Failure of Circulation.—Starling, in 1912, found that with a blood-pressure below 80 the cardiac output is diminished. This is due to the diminished rate of capillary circulation in the cardiac muscle. The cells of the whole body suffer in the same way owing to deficient intracellular oxidation.

It may be argued that the changes described in the cells of the central nervous system in fatal cases of shock are not the cause, but the result of this process. Cannon regards a blood-pressure of 80 as the critical level. When the pressure remains below this level for more than four hours, the patient will not recover, no matter how heroic are the measures of treatment employed.

The diminution of intracellular oxygenation, which is associated with the low body temperatures found in shock, is now combated by the injection of insulin, which will be referred to later.

Older Theories as to the Circulatory Failure in Shock.—The "splanchnic pool" no longer exists. Observed facts have supplanted fantastic hypotheses, and such methods of treatment as the pneumatic suit or raising the foot of the bed are now obsolete.

VI.—*Toxæmic Factors in Shock.*—Clinical evidence for the support of the adrenalin theory has already been produced.

Experimentally, Cannon and also Elliott have demonstrated the presence of adrenalin in the circulating blood of animals under emotional stress. Bedford has

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been able to show the presence of adrenalin in the blood of animals suffering from experimental shock. Bainbridge and Trevan found that intravenous injection of small doses of adrenalin into an animal induces, after twenty minutes, the concentration of blood already described.

Dale's "Histamine Shock" opens up a large field of thought. Dale and his co-workers found that suitable doses of histamine (10 mgm. for a large cat) produce a profound fall of blood-pressure, with capillary stasis and subsequent concentration of the circulating bloods. Smaller doses cause a vaso-dilatation, and evidence is produced to show that this is capillary in origin. This work deserves consideration in conjunction with Bayliss's investigations on muscle trauma. Bayliss and Cannon found that within an hour after producing a compound fracture of the femur in an anæsthetized cat, signs appeared similar to those seen in secondary wound shock. The pressure gradually went down, pulse-rate and respiration increased, the blood became concentrated and finally the animal died. This occurred just as rapidly when the limb was isolated from the central nervous system; so that the possibility of its being due to the transmission of harmful afferent stimuli was negatived. When, however, the returning blood-stream was interrupted, no lowering of the pressure resulted, and the animal remained in good condition until the clips on the vessels were removed. As soon as the returning blood from the traumatized area reached the general circulation, down came the blood-pressure. It is probable that some tissue poison is set free from the traumatic myolysis which has resulted, producing effects like histamine.

The importance of the toxæmic factor in shock is now becoming more widely recognized than ever before. Several Continental workers have published works on this subject. Cornioley and Kotzareff [9] in 1921 found that (*a*) the more marked the anatomical lesion the more prolonged is the shock, and (*b*) the more massive the toxæmia the shorter is the incidence of shock. Quénu [10] found that blood taken from a shocked animal always induces shock when injected into a fresh one, producing rigors, lowering of body temperature, coma, etc. In further experiments these workers claim to have prevented shock by injecting antibodies produced by injecting animals with small doses of shocked blood.

On the clinical side it is stated that the phenomena of toxic shock arise in the following order of frequency: (1) Fractures of bones; (2) burns; (3) fatigue; (4) malignant tumours. Quénu [11] from his clinical studies also classifies shock into "primary" and "secondary." De Courcy [12], in 1922, accepts the idea of secondary shock and considers those reactions that follow in from twelve to twenty-four hours as coming under this category. He thinks that the main factor is the absorption of toxins produced by destruction of tissues in rough manipulation. To combat this effect he attempts to desensitize the patient before operation by injecting typhoid vaccine, and claims success for this method.

Further proof of toxæmia in shock has been obtained experimentally by taking two animals and crossing the circulation. Induction of shock in one animal leads to a corresponding fall of blood-pressure in the other. (McIver and Haggart [13].) On the clinical side there is ample evidence in favour of the toxic factor in shock. Several cases have already been quoted in which the presence of toxæmia, either tissue or bacterial in origin, has been the potent cause of shock. If this absorption of toxin idea is true, a clear indication is afforded as to the prevention of shock in surgical procedures. This is well illustrated by the following case.

Two years ago I performed a fore-quarter amputation of the right upper limb in a woman aged 72 for sarcoma of the upper end of the humerus. The main vessels were exposed and clamped at the commencement of the operation. The unblocked brachial plexus was left intact, and was undivided until half an hour later, when the limb was finally removed. The blood pressure remained constant, and no shock developed subsequently.

Afferent impulses may therefore be considered harmless so long as the higher nerve centres are cut off, as in surgical anæsthesia. Pain itself produces a fall in blood-pressure. Reference to this chart shows the fall in blood-pressure from 135 to 65 in about an hour and a half during attacks of renal colic. When the pain was relieved by morphia the pressure quickly rose to normal. Bacterial toxæmia supervening within a few hours after a wound or surgical operation exerts a profound influence on the blood-pressure with the establishment of secondary shock. If an unsuitable anæsthetic is employed a chemical toxæmia may be added to the shock causation complex, with serious results.

Chloroform, and to a lesser extent ether, are powerful toxic agents. Buckmaster pointed out many years ago that the presence of chloroform in the blood greatly diminished the oxygen-carrying power of the red corpuscles, thus aggravating the already present deficient tissue oxygenation.

M. K. Cattell [14], in 1923, working in Cannon's laboratory, published the results of extensive studies of the blood-pressure in ether anæsthesia. Some of the more important conclusions are:—

(1) In the normal animal strong ether inhalation results in a sudden temporary drop of blood-pressure. The pressure recovers quickly, so that it is normal by the time the eye reflex has gone.

(2) In the shocked animal there is no recovery after the primary fall. The pressure continues to fall, even to zero, as the anæsthesia is continued.

(3) Nitrous oxide and oxygen can be given to a shocked animal with only a minimum drop in the blood-pressure.

(4) (No. 9 on Cattell's list.) Ether sensitiveness is induced by: (1) Low blood-pressure. (2) Hæmorrhage. (3) Severe operations. (4) Injection of acid.

These observations on ether and shock confirm the opinion surgeons now hold on the value of gas and oxygen anæsthesia. In shock and severe operations where shock is expected to occur, it is absolutely unjustifiable to give any anæsthetic other than gas and oxygen.

Recognition of the tissue toxæmia occurring in shock establishes the relationship of surgical shock to anaphylactic shock.

It is convenient here to discuss the effect of morphia in shock as an additional toxic factor. The beneficial effect of small doses of morphia, which may be repeated, has already been shown. Large doses slow respiration, produce cyanosis, lessen the intake of oxygen, and diminish the already failing intracellular oxygenation of the tissues. At one time carbon dioxide was thought to be beneficial in shock (Acapnia theory). Cyanosis should always be regarded as a dangerous condition and avoided or actively combated on all occasions.

VII.—*The Central Nervous System.*—The earlier views on the pathology of shock all centred round the nervous system. Exhaustion of the vasomotor centre, fatigue of the higher centres, peripheral vaso-constriction and splanchnic dilatation were each in turn discussed as the essential factor.

Little will be said here of the nervous side of the subject. From the practical point of view, excitement, fatigue and anxiety must be dealt with, and steps taken to relieve pain. Otherwise the nervous theories of shock may be kept for academic and historical discussion only.

VIII.—*Metabolism in Shock.*—In this connexion the work of D. Fisher [15], of Boston, is interesting and of great practical importance. He classifies shock into four groups: (1) Traumatic; (2) septic or toxic; (3) anaphylactic; (4) nervous.

Studies of the blood-sugar after a Marathon race showed that it is normal when no distress is present. In one runner, who fell unconscious, a low blood-sugar

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content was found—the picture of an overdose of insulin. Fisher conceives shock as a condition in which there is an internal asphyxia and acidosis with oxidative processes held in abeyance, producing the resulting exhaustion. Any method of promoting combustion and oxidation, and at the same time furnishing heat energy, should be effective. Therefore, to combat shock rationally, give a substance that will give rise to an immediate supply of energy, maintain it, and at the same time keep up the circulating volume. For this purpose insulin is used, combined with hypertonic glucose solution. The following cases of Fisher's are quoted as examples of this treatment.

(I) *Surgical Shock.*—H. A., male, aged 25, was submitted to an arthrodesis of the shoulder-joint for tubercular disease. The operation lasted two and a half hours. His pulse increased from 96 to 150, the respiration from 18 to 35, and his blood-pressure fell to 75. On return to bed he was pulseless with respirations of 40. A litre of 10 per cent. glucose was slowly given intravenously. Insulin was injected subcutaneously at the beginning and end of the infusion, 25 units each time. The reaction was almost miraculous. Two hours later the pulse was 116, respiration 22, and blood-pressure 112. Acetonuria was present, but had disappeared eight hours later.

(II) *Toxic Shock.*—A woman, aged 64, became shocked twenty-four hours after an operation for a gangrenous gall-bladder. Her temperature was 104° F., pulse 140, and respirations 36. A litre of glucose was given slowly for two and a quarter hours with insulin, as in Case I. Six hours later her pulse was 108, temperature 102° F., and respirations 22, and her subsequent convalescence was straightforward.

Other cases are quoted and other observers have obtained similar results. Fisher states that there is no danger as long as there is glucose in the urine. To counteract an insulin reaction, the juice of an orange, or cane sugar, may be given with or without a hypodermic injection of adrenalin. He also advises that rectal saline should be given at the same time.

Acidosis was at one time considered an important pathological factor. It is now recognized as the result and not the cause of a low arterial level. Acidosis disappears as the pressure rises.

IX.—*Present Conception of the Pathology of Shock.*—Shock may be divided into primary and secondary varieties. Initiating factors are: (1) Pain; (2) hæmorrhage; (3) cold; (4) toxæmia, tissue, bacteria; (5) anaphylaxis.

Sustaining factors are: (1) The low blood-pressure; (2) the decreasing blood-volume.

The results produced are: (1) Diminished circulating fluid; (2) deficient intra-cellular oxygenation; (3) devitalization of important organs as heart, brain, kidneys, etc.

X.—*Treatment.*—The more exact our knowledge becomes concerning a given pathological condition, the simpler and more successful is the treatment. The conception of secondary shock led to the adoption of measures for its prevention. Such front line measures, which are published in the Army Medical Manuals, and taught to-day in the R.A.M.C., are familiar to all present. In surgical centres the importance of the organization of "pre-operation" and "resuscitation" wards is admitted by all. In civilian work to-day the same principles of efficient first aid and splinting, administration of warmth and fluids and rapid evacuation to hospital are being taught everywhere.

In the operating theatre, shock may be avoided by warmth, choice of suitable anæsthetic, i.e., local, spinal, or for a bad case gas and oxygen, hæmostasis and gentleness in handling the tissues.

Starving the patient and withholding fluids too long beforehand tend to produce anhydræmia and should be avoided. A small dose of morphia may allay

mental pre-operative apprehension. In the wards the same general principles again apply. If shock develops and a blood-pressure of 80 does not respond to simple measures within half an hour, then give a litre of 10 per cent. glucose saline, with insulin at the beginning and end of the infusion. Even after a severe hæmorrhage this will tide the patient over the crisis, and infusion of whole blood (transfusion) may be done subsequently if necessary.

The question of operating in shock must be decided in each case. If a suitable anæsthetic is employed, surgery may be the only means of removing the cause of toxæmia and thereby saving the patient.

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Discussion.—Mr. ZACHARY COPE said that he thought the distinction made by Lieutenant-Colonel Cowell between primary and secondary forms of shock was sound and useful, but if it was accepted certain corollaries followed. The term "shock" must be widened to include similar conditions due to dissimilar causes. The term "primary shock," which included the immediate effect of severe wounds, should be allowed to embrace the result of external burns or internal visceral ruptures, whilst "secondary shock," which was probably due to toxins absorbed from injured tissues, must include those similar states of extreme bodily depression following the absorption of toxins from the alimentary canal in late cases of intestinal obstruction.

The condition of depression which sometimes followed immediately after the perforation of a gastric ulcer was indistinguishable from shock and, though one might apply the term "collapse" or "exhaustion" or "prostration" to it, clinically it was shock.

A rapid pulse-rate was not an invariable accompaniment of shock, for Lieutenant-Colonel Cowell had published details of the most extreme shock with a slow pulse-rate; neither was a low blood-pressure a necessity of the shocked condition. The essential circulatory disturbance was a diminution of the volume of blood in circulation, and this might occur without any appreciable lowering of blood-pressure. Of course, in the last stages, the blood-pressure rapidly fell. It was strange that the blood-pressure was relied upon so much when, as a rule, the patient's normal blood-pressure was unknown and therefore the amount of lowering could only be guessed at. Moreover, as a rule, only the systolic pressure was relied upon, whereas it was more important to note the difference between the systolic and diastolic pressures, i.e., the pulse-pressure.

Sir MATTHEW FELL said that in the early stages of the Great War the administrative policy was to evacuate all cases down the lines of communication, but that later, behind a fixed trench line, a progressive forward development of operating facilities had taken place. He wondered whether, from the point of view of shock, the latter policy would have been the wisest from the beginning of the war, or whether it was applicable to a mechanized force which might be operating one-hundred miles from its railhead and casualty clearing station.

Dr. KINGSTON BARTON said that Lieutenant-Colonel Cowell had clearly advised the use of glucose, with normal saline and insulin, both as a preventive of secondary shock and for its treatment when established. It would interest him (Dr. Barton) to know whether alcohol, given as a remedy for primary shock, might also help to diminish secondary shock. He could understand that when secondary shock was established, especially if due to toxæmia, then the use of alcohol might not be a wise procedure. Throughout his professional life he had strongly held

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that alcohol was detrimental to people in normal health, especially to those aged under 21 years. But this did not prevent him from realizing the great value of alcohol (brandy for choice) in many cases of sudden collapse, medical or surgical. So that he wondered whether modern surgeons placed any value on the administration of alcohol before any severe operation, in which shock might be expected, in order to prevent or diminish such shock. Also, whether a dose of alcohol was of any use when a patient, civil or military, was brought in in a state of primary shock.

He remembered how, in the early days of anæsthetics at St. Bartholomew's Hospital, Mr. Luther Holden used to order, with apparent advantage, a glass of wine or brandy for any patient who looked pale or collapsed before coming into the operating theatre. With regard to the recent reliance on doses of glucose we must remember that fifty years ago the French travellers and the physiologists always advocated heavy doses of sugar in hot water as the best restoratives in cases of collapse or exhaustion, or of severe injuries.

Dr. HALLS DALLY said he regretted a recent tendency to minimize the significance of low arterial pressure in relation to shock. It might be that shock in the early stages of its less acute and grave forms could develop, unaccompanied by any appreciable drop in arterial pressure, but, before accepting such view, he would require definite clinical evidence. He was not aware of any authenticated case in which a late stage of shock had existed, with a well-maintained pressure, as suggested by Mr. Zachary Cope at a recent meeting of the Section of Surgery,[1] and could hardly believe it possible. From his own view, which he regarded as fundamental, that hypopiesis, however caused, was invariably an expression of a low vitality state, of which shock forms a typical example, it was not difficult to see why hypopiesis should occur as a usual accompaniment, although it was possible that initial vaso-constriction might for a short time, and under certain conditions, resist this fall, but even then not as a constant factor. He urged that serial investigations should be made on cases of shock, with accurate instruments, recording both diastolic and systolic pressure. Since many fallacious deductions had been made, thanks to estimation solely of the systolic pressure and to other faults in estimation.

1 *Proceedings*, 1928, xxi (Sect. Surg.), 599.

Section of Odontology.

President—Mr. W. H. Rushton, L.D.S.E.

[*January* 23, 1928.]

CASUAL COMMUNICATION.

Case of Necrosis of the Mandible.

By H. Paxton Bayliss, L.R.C.P., M.R.C.S., L.D.S.

The patient was aged 42, and had been treated for twenty years for a syphilitic infection. In November, 1926, he was having intermuscular mercurial injections, and intravenous novarsenobillon. He developed diarrhœa and gastric symptoms, and the lower six front teeth, his only remaining ones, were extracted under a general nitrous oxide anæsthetic. The extractions were followed by extensive ulceration of the mouth, and he was taken off the mercurial and novarsenobillon treatment, and given fairly large doses of potassium iodide. The surface of the bone of the anterior portion of the mandible then became denuded of soft tissues and he lost sensitiveness in his lower lip. In March, 1927, he presented himself at the Dental Department of Guy's Hospital with almost the entire depth of the mandible exposed to the extent of the sockets of the six front teeth. He continued to use a mouth-wash of liquor potassæ and carbolic acid and he kept the exposed bone clean by swabbing it with hydrogen peroxide. Six months from the time of the extractions the sequestrum was sufficiently free to be lifted out, and six weeks later he was satisfactorily fitted with a lower denture. Sensation subsequently returned to his lower lips.

[*February* 27, 1928.]

Dentistry as One of the Fine Arts.

By Lilian Lindsay, L.D.S.Edin.

[ABRIDGED.]

In basing my paper on the remarkable work of Girdwood[1] on "Tube Teeth and Porcelain Rods," I am not concerned either with the utility or the desirability of crown and bridge work in the mouth, or in argument on the use of these agents in dental prosthesis, but to advocate the use of "tubes" and "rods" as a means of training and developing the artistic faculty of the dental student by setting him to work on a medium to be associated with his practice, the beauty and possibilities of which ought to appeal to his creative instinct. As the student in sculpture must acquire an intimate knowledge of surface anatomy, so the dental student ought first to make an accurate study of the minute surface anatomy of the human tooth, and of as many of the different forms and shapes thereof as can be shown. The student must learn also to arrange teeth in proper occlusion, working from the natural teeth and not from porcelain imitations, and here I suggest that an essential subject of the preliminary examination should be freehand drawing. Professor Tonks, in a paper read to the Eastern Counties Branch of the British Dental Association in 1926,

[1] "Tube Teeth and Porcelain Rods," John Girdwood, 1918.

pointed out that although the dentist is responsible for restoring the true contour of the human face, he is not taught to study "contour" or to appreciate "solidity"; he looks upon the human countenance as if it were in two dimensions, the third dimension—depth—being left out of account. This third dimension is not only essential to contour, but also seriously concerns phonetics and mastication. From the introduction of the flat porcelain tooth by Fonzi in 1808, dentistry started on a stereotyped progress which, aided by the later introduction of vulcanite, from an artistic point of view, degraded it.

By his inspiration to make dentures in porcelain, Dubois de Chemant made the first attempt to rid humanity of the abominations of bone and ivory which, notwithstanding the most scrupulous care, could only with difficulty be prevented from being an offence to the public and a danger to the health of the wearer. It is interesting to recall the methods by which porcelain was first adapted for use in artificial dentures. The material, made into a stiff paste, was moulded to the model, and allowed to dry and harden until the teeth could be carved in the lump with sculptors. The piece was then fired, and when cold was placed on the model, which was moistened, sprinkled over with red oxide of lead to test the inequalities in the set, and any necessary correction made by grinding with wheels. The enamel was then applied and the piece once more fired. This porcelain denture, while not always all that could be desired, had great possibilities in skilful hands, and was revolutionary in its effect on oral cleanliness; contemporary literature shows that it attracted the attention of the more advanced practitioners of dentistry. Unfortunately the introduction of the single flat tooth by Fonzi gave the unhealthy bone base another half century of existence.

Some time after the introduction of Fonzi's flat tooth there was invented in this country another type of tooth which offered a wide range of artistic use. Corbett, of Cork, in his Presidential Address to the British Dental Association in 1888, describes how his father and a Mr. Hallit had worked together sixty years to produce the tube tooth, and his story of their adventures is scarcely less thrilling than that of a Pallissy or a Cellini. The tube tooth fulfils the requirements of solidity and contour. It is more natural in appearance and stronger than the flat tooth, and it has possibilities of extended usefulness and adaptability which were largely unappreciated until within the last twenty years they appealed to a modern dentist who had used the tube tooth and its derivative the porcelain rod, to obtain every variety of tooth and combination of teeth. Dealing first with the single tube tooth, the slide shows how a central may be shaped from a molar in three minutes, also the shaping of a central, a lateral, or a canine, from an extra large bicuspid in a minute and a half, thus emphasizing the adaptability of the tube tooth [slide shown]. It is asked whether it is possible to re-polish porcelain teeth which have been cut or from which the superficial glaze has been removed. Certainly it is, especially with what are known, or used to be known, as "English teeth." Tube teeth and the porcelain rods with which we are dealing have a homogeneous texture which lends itself to perfect polishing. In manufacture these teeth are "poured," that is, they are made at one firing, body and glaze combined, as opposed to the ordinary type of flat teeth which are moulded, body and glaze taking two bakings, and which, when the surface has been removed, show porosity which interferes with polishing. Added to their handiness in use and their "third dimension" qualities, the translucency of the tube tooth is not spoiled, as is largely the case with backed flat teeth when used in plate work, nor is there that risk of cracking or displacement during soldering to which the backed platinum pin flat tooth is exposed. Another important point which is not appreciated as it ought to be is the "feel" of these teeth to the tongue in speaking; even the "anatomically" formed molars and bicuspids are frequently

too small in the crown for the perfect utterance of various important sounds. These are a few of the virtues of the tube tooth, and they enforce my point that the use of a perfect agent in the mechanical training of the dental student could not fail to help him in his future work.

The possibilities of artistic training of the student are extended and improved by the use of the single or double tube porcelain rod, which is simply the tube tooth extended into a wider sphere of usefulness. These figures show various sizes and shapes of these rods [slide shown]. The holes which penetrate the blocks for vertical use like the ordinary pin hole in a single tube tooth, or horizontal use, are meant to permit the attachment of the finished and carved block of two, three, or fourth teeth to the pins, crowns, gold inlays, or bars, which are to retain the work in position in the mouth.

The first thing to be taught is the exact surface anatomy of the tooth ; for this purpose natural teeth may be mounted in plaster, and should always be in front of the worker until he becomes an adept, for it is the knowledge of where to cut which makes the shaping of a tooth easy and quick, whether from a tube or from a porcelain rod. The next step is to outline the teeth on the rod in pencil as a guide to subsequent cutting [slide shown] ; and here is the importance of instruction in drawing as an essential of the preliminary education of the dental student. The next figure shows the method of defining the cervical outline and a grinding wheel used in the process. Skill in grinding the natural shapes of the teeth accurately and rapidly depends chiefly on a knowledge of their surface anatomy and the practice of first outlining them on paper and then on the porcelain rods from which the glaze has been removed. These points cannot be over-emphasized, since only proficiency in them gives the requisite knowledge, amounting to intuition, of where to grind. This is half the battle in conquering the dislike of it, which gives a false impression that this work is arduous and a waste of time. Shaping does not consist in grinding off large portions of porcelain, but in knowing exactly where to grind. A point scarcely less important is the use of the right kind of grinding wheel. The slide [shown] illustrates the amount ground from a tube-rod in one minute with a carborundum wheel, size 4 in. by $\frac{3}{8}$ in., grade K, grit 80, vitrified bond, speed 1,200 revolutions. The edge of the cut is rough and chipped in contrast to the other edge, ground with an elastic bond wheel, same size and speed, but grit 90, which is clear and smooth. The manner of holding the rod to the wheel is also important, and the position of the hands is no less so.

While using material which responds to artistic impulse, the student is gaining familiarity with and mastery over one of his most important pieces of equipment. the dental engine. The grinding wheels are usually fitted for use in its handpiece, and this continuous use of the engine should introduce the student to those fascinating problems of the grits and bonding materials most suitable for abrasive wheels and to the study of the effects of pressure and of speed in obtaining the maximum cutting results from all kinds of revolving cutting tools, as set forth for example in " A Propos de nos Fraises," by Emile Huet.

I show specimens, not as advocating at present their use in dental prosthetics, although that can be strongly supported ; my purpose is to stress the value of this method in training the hand, the eye and the imagination of the student. This side of dental training has suffered in the last twenty-five years and has diverged widely from the path designed for it in 1842, when John Gray postulated the two essentials for the practice of dentistry, namely, knowledge of pathology and surgery, and knowledge of that which concerns the nice construction of artificial teeth.

As long as carving in bone and ivory continued, there was scope for the

development of manual dexterity and of the imagination, in a way less perfect than is possible with porcelain, but still valuable. The introduction of vulcanite as a base, synchronous with a like mechanizing development in other fields, led to deterioration, and the dental profession turned towards the easier paths of theoretical science, resulting in the decrease of mechanical study and a tendency to treat it as of diminishing importance. The number of theoretical subjects which the student now has to master may seem to warrant the alterations referred to, but a closer knowledge and estimation of the part which the dentist has to play will prove that the dentist, no less than the surgeon, is concerned mostly with the handling of instruments and that his training should be directed accordingly. Fortunately some departments of original work on scientific dentistry demand an even higher manual dexterity and a more delicate skill than some of those on the practical side, and thus the line of training I suggest will assist the student to make the best both of research and of practice. I do not say there is lack of artistic skill in the profession to-day—the exhibition of hobbies at the Liverpool meeting of the British Dental Association would itself give the lie to such a suggestion. I am only indicating a way in which the training of the future student may be improved, and any incentive to creative effort in the present system increased.

We may hope that under the new art it will be impossible to tell whether teeth are natural or artificial, and that when the present propaganda in oral hygiene has perfected and completed its good work, the arduous task of conservation and repair will be lightened, both for the operator and for the patient, by dexterity of touch guided and controlled by that delicacy of feeling which I believe would follow from a mechanical training in which a large part was taken by such exercises as I have endeavoured to explain and to illustrate in the carving of tube teeth and porcelain rods.

[The paper was illustrated by a large number of lantern slides and specimens.]

CASUAL COMMUNICATION.

A Case of Necrosis of the Mandible and Exfoliation of the Condyle and Coronoid Process following Suppuration in the Region of the Third Mandibular Molar.

By A. T. MARSTON, L.D.S.

THIS case occurred in private practice. The history is as follows. In February, 1923, the patient, a man, aged about 36, consulted a medical man on account of an abscess which had developed about his third right mandibular molar, and the patient told me that he was advised not to have the tooth extracted while the swelling was there, and that he was told to apply hot fomentations to the outside of the face. This treatment was followed. After four weeks in bed, and a further four weeks at the seaside to tone him up, followed by a further three weeks in bed, the patient's condition was brought to my notice on May 11. His face was very swollen; there were sinuses opening externally about the angle of the jaw; there was trismus but still the incisor teeth could be separated about three-quarters of an inch; and on the inside of the mouth, the third right lower molar was very loose and was bathed in pus, and pus was discharging from around the $\frac{7}{54}$. There were also sinuses through the

mucous membrane distal to the third molar. The immediate extraction of four teeth was performed under continuous nitrous oxide without difficulty, and a longitudinal incision made to join up the sinuses to permit of drainage. A large quantity of pus came away. The wound was well syringed with a solution of sodium bicarbonate followed by dilute hydrogen peroxide to which a little tincture of iodine was added. A 3-oz. rubber syringe with detachable nozzle was provided the patient, and as it was obvious that the pus had riddled the bone, and constant irrigation would be necessary for some months, while Nature was dealing with the sequestrum, his wife was shown how to carry out the syringing three times a day. Eusol or Dakin-Carrel solution was prescribed as the irrigant. By the third day the external sinuses had ceased to discharge and it was noticed then that a probe could be passed up on the outer side of the ascending ramus. The subsequent treatment resolved itself into waiting for the sequestra to exfoliate, the patient coming each week to enable me to thoroughly syringe with the bicarbonate, and with the peroxide and iodine. Gradually the exfoliation rose above the gum, and to such an extent that they gagged the bite. Yet knowing that separation was not complete, I twice removed smaller extrusions using excising forceps. It caused less discomfort to thus nip off the softening necrosis than would have been caused by cutting it through with the dental engine. In October, wishing to ascertain how much the ascending ramus was affected, I persuaded the patient to have the part radiographed, which he did at a general hospital. I was not able to procure the plate, but received the following report, October 22 :—

"There is an extensive sequestrum, involving about half the vertical thickness of the right side of the mandible in its horizontal part, but not affecting the ascending ramus. The sequestrum has the appearance of being detached, with a clear area, presumably occupied by pus and granulation tissue, between itself and the remainder of the jawbone. There is no involucrum to be seen at present. The dead piece seems to be exfoliating slowly, but satisfactorily, and unless the man is tired of it, or the natural process of cure seems at any time to be hanging fire, I think we might as well let Nature go her own way to work."

On November 9, the exfoliation being projected upward and forward, and interfering with the free closing of the mouth, the anterior portion above the gum was separated under continuous nitrous oxide, using the dental engine. The posterior portion was still attached. On March 11 the exfoliation was complete, and again under continuous nitrous oxide, the sequestrum comprising the whole condylar process was brought away with bayonet forceps, with a plain rotary movement. In the following August the swelling due to the callus had cleared up, and when I saw the patient in the following February he assured me that he experienced no difficulties from the side which had been affected. Besides the exfoliation of the condyle, it is of interest to note that masticatory movements were very little impaired throughout, that the fresh bone was re-formed while there was no period of rest, that the nerve-supply to the remaining anterior teeth was unaffected, and that the period for the complete exfoliation was twelve months. The idea of using the alkaline irrigant before the disinfectant was to saponify the fatty degenerative matter covering the lesion, thus permitting the disinfectant to reach the spot, and by the addition of the iodine to the peroxide, nascent iodine is liberated, and being nascent is more effective. Cases of exfoliation of condyles, with greater or less portions of the body of the mandible, have been reported after exanthemata, fracture of the neck of the condyle, and also following arsenical necrosis, and in the reports I have read there seems to have been little interference with masticatory movement.

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[*April* 23, 1928.]

CASUAL COMMUNICATIONS.

Injection of Alcohol for Neuralgia: and its Sequel.

By W. H. RUSHTON, L.D.S. (President).

A CLERGYMAN aged 69, edentulous, consulted me five years ago for persistent and acute facial neuralgia on the left side. As radiographs had shown nothing abnormal I sent the case to Mr. Wilfred Harris who injected alcohol into the left foramen ovale with complete success, the patient having had no recurrence of the tic. Some twelve months later he came back with his lower denture no longer articulating with his upper. When the jaws were approximated the lower bite had shifted about half-an-inch to the right of the corresponding upper; while when he opened his mouth the mandible descended to the left. I re-made the lower denture to the altered bite and his mouth has remained quite comfortable until recently when the bite has had to be readjusted. Mr. Wilfred Harris sends the following note on the case :—

"In injecting the left foramen ovale, the motor root of the fifth nerve is always paralysed together with the sensory fibres, so that besides weakness of the masseter and temporal muscles for the up-and-down bite, the external pterygoid is also paralysed, consequently in opening the mouth, the lower jaw drops towards the left side, being pushed over by the sound external pterygoid of the other side. In closing the jaw to bite, the sound internal pterygoid and masseter and temporal will pull the mandible slightly too far across (to the right). Usually the motor root recovers very considerably, even when the ganglion is injected, as its trophic cells lie further back in the pons, but in some cases the muscles do not recover, and then there is obvious wasting above and below the zygoma, which stands out prominently."

It would be interesting to know whether, if the patient had not been edentulous, the same result would have occurred or whether the interlocking of the cusps of the opposing natural teeth would have been sufficient to maintain the normal bite.

A Paste for Obtunding Sensitive Dentine.

By J. LEWIN PAYNE, L.R.C.P., M.R.C.S., L.D.S.

A SIMPLE means of desensitizing dentine without endangering the health of the pulp, or the periodontal membrane, is a procedure which all who practise conservative dentistry desire. The use of a local anæsthetic injected into the soft tissues, or the bone, will allay pain during the time of preparation and filling of the cavity, but the dentine remains sensitive after the effect of the anæsthetic has passed off, and, in some cases so treated, it continues to be irritated by the contact of even comparatively bland filling material. Moreover we all meet with many cases of sensitive areas at the necks of teeth due to recession at the gum margin, and to abrasion or erosion. In each of these some agent is needed which will remove the sensitiveness of the dentine without damaging the pulp or periodontal membrane. Many drugs have been used for this purpose, but most of them have proved unsatisfactory, either because they do not permanently desensitize, or because the desensitization goes too deep, and ultimately causes degeneration of the pulp. In a communication of this nature, time will not permit me to refer to the comparative merits of the drugs so employed, but I desire to press the claims of one drug which was formerly employed for this purpose. The drug I refer to is paraform, a polymer

of paraldehyde. From fifteen to twenty years ago paraform was used by many practitioners for obtunding sensitive dentine, and Dr. Buckley suggested a proprietary paste containing some of this drug. But, later, it was generally condemned and discarded by most practitioners, for the reason that intense irritation, injury and—in many cases, death—of the pulp resulted from its use. I am now pleading for a reconsideration of this drug on the grounds that, in former days, it was commonly used improperly. Like many other potent and valuable drugs, if given in large doses it acts as a poison, and the real medicinal value is lost. For the past ten months I have been testing various preparations, and I find that almost incredibly small doses of paraform, used in suitable combination with other materials, produce the best results. I am indebted to Mr. E. Williamson, the pharmacist to Guy's Hospital, for help in making these experiments. Experience has shown that doses of $\frac{1}{300}$ to $\frac{1}{100}$ of a grain of paraform in suitable combination with orthoform, thymol, wool and paraffin will give excellent results. A paste sufficiently coloured to be seen when in contact with the dentine serves best. The preparation of paraform paste is composed of:—

Paraform	...	...	...	3 parts
Orthoform	...	...	...	2 ,,
Thymol	...	...	...	0·25 ,,
Wool and paraffin		...	...	1·75 ,,
Soft paraffin	...	...	...	7 ,,
				14

Thus the paraform represents roughly one-fifth of the whole mass. It is essential that the ingredients shall be very carefully mixed. As is well known *paraform* acts by giving off formaldehyde vapour at the temperature of the body. It is not only a powerful antiseptic capable of destroying micro-organisms on, and just beneath, the surface of the dentine; but it has also a marked effect in hardening soft tissues with which it comes into contact. *Orthoform* has anæsthetic properties, and produces the reddish-brown discoloration of the paste which is so useful when making the application on a yellowish-white tissue like dentine. *Thymol* is a non-irritating antiseptic.

The quantity of this paste required for desensitizing a small cavity need not exceed in bulk a piece of the paste the size of a pin's head; by that I mean the head of the ordinary pin used for domestic purposes. Sometimes even less will suffice. For an average cavity the total weight will be about ·001 of a grm. The amount of paraform being ·0002 grm., which represents roughly $\frac{1}{300}$ gr. For carious cavities only the soft superficial caries need be removed. Then, after cleansing the surface with iodine and absolute alcohol, smear the paste very thinly over the surface to be desensitized, cover with a few shreds of cotton wool and carefully seal with plastic cement-filling. For abrasion and erosion cavities at the neck of a tooth, tartar and food débris, if present, must be removed. The surface is cleansed with alcohol, and dehydrated. The gum may be protected by means of an ether varnish. Then smear a tiny piece of the paraform paste over the area to be treated and seal it in with a sticky oxyphosphate cement giving time for the cement to set well against the smooth surface of the tooth, before the saliva is allowed to come into contact with it. Two or three teeth can be prepared in this way in the course of a few minutes. In each case the dressing is removed within forty-eight hours. One day is often sufficient for the purpose. If desensitization is not complete after one application, the treatment may be repeated. I have used this attenuated form of paraform in more than two hundred cases, and, in no instance, when it has been correctly applied, has there been any evidence of pulp death, or degeneration. In about 10 per cent. of the cases, patients complained of slight pain during the first

twenty-four hours. This pain, however, when dealt with promptly, can be quickly relieved by means of sedative drugs. If pain does occur after the application the patient should be asked to return within twenty-four hours for the removal of the dressing.

I must conclude with the warning that paraform is useless as an obtundent in cases in which only a very thin layer of dentine lies over the pulp. It is dangerous to the pulp if large doses are employed, but, if it is used in the attenuated form that I have mentioned, it will prove an every-day boon in the treatment of sensitive dentine.

[*May* 21, 1928.]

A Description of Some Specimens Illustrating the Pathology of the Teeth of Elephants.

By Sir FRANK COLYER, K.B.E., F.R.C.S.

DURING the month of January, 1926, several letters on four-tusked elephants were published in *The Times*. One writer, Capt. Tracy Phillipps, stated that he stayed a night at the residence of the Vongara-Zande Sultan Ngilima, who described how he had killed a "four-tusker" during the war. Capt. Tracy Phillipps himself while hunting on the Congo-Nile Divide, roughly where 28 deg. E. long. cuts the frontier, came upon a small herd of elephant about forty in number and one of the natives shouted that there was a Siyāmma with the herd. The animal was an "enormously out-standing beast, which made all the others look relatively very small." Directly danger was scented the rest of the herd at once drew closely round him and shouldered him off with extraordinary rapidity and care. In one letter reference was made to an elephant with four tusks seen by an army officer in the region of the Congo-Divide. The account published in *Sudan Notes and Records* (Vol. II, 1919, p. 231) is as follows:

"On May 18, 1917, I went out shooting in the district of Sheikh Ako Mangara, in the Markaz of Yambio, in the village of Wakila Marbo, on the borders between Tembura and Yambio districts. I met a herd of elephant, which I followed, searching for a good one to shoot. I kept following them until they stopped near a pool of water, where they began to drink and throw mud on themselves. I was hiding behind a tree about 15 yards from them, looking at them, when I saw an elephant with four tusks. The left tusk was the bigger and had the usual direction, but the direction of the small tusk was downwards and came out from under the big one. It was round, and its thickness was about 2½ in. The direction of the right tusk was downwards, and the small tusk came out from under it in the usual direction, but it was small, like the other one. Abd-el-Farag (Mulazim Awal)."

It is of interest to note that the natives of the district do not question the existence of four-tusk elephants. I do not know of an elephant skull with four tusks. We have specimens of double tusks on one side and from our knowledge of anatomical variations there is reason to assume that the variation may be symmetrical. An example of a double tusk is shown in fig. 1. The two portions are of unequal size, and where the surfaces are in contact each portion is somewhat S-shaped.

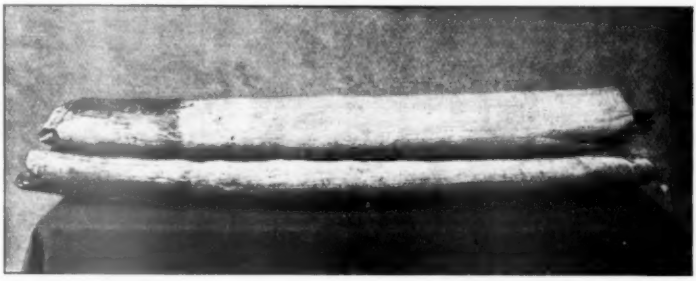

FIG. 1.—An example of a double tusk. Roy. Col. Surg. (Odonto. Series), G120.01.

FIG. 2.—A specimen showing a stage in the production of a double tusk. Roy. Col. Surg. (Teratological Series), 540.

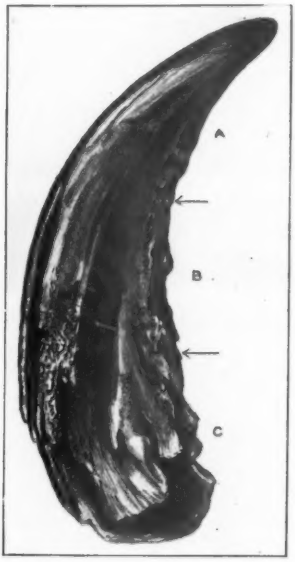

FIG. 3.—A tusk which has been injured. Roy. Col. Surg. (Odontology Series) G122.6.

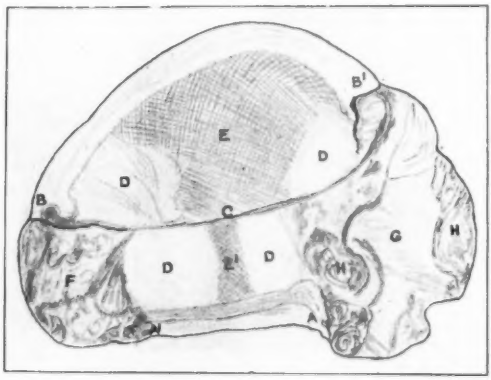

FIG. 4.—Diagrammatic view of a transverse section of the tusk shown in fig. 3.

The specimen (fig. 2) seems to show that the double tusk is at times due to dichotomy of the tooth-germ; in this tusk there are definite grooves on opposite sides of the tusk.

An example of a double tusk was brought to my notice by a big game hunter in East Africa. One tusk weighed 27 lb. and was twisted so that it curved downwards and backwards bringing the point between the elephant's knees. At the base of this tusk was another small tusk the end of which had been broken. A piece of the surrounding bone was present so that one was able to obtain an accurate idea of the relation of the one tusk to the other.

We are fortunate in possessing several specimens illustrating the great recuperative power of the tusks following severe injury. I wish to refer to four specimens which have been added to the collection during the past three years. The tusk (fig. 3) is about 95 cm. long as measured on the outer curve. The tusk is compressed on one side, the compression deepening towards the terminal portion. The tusk was divided into three sections by two cuts made, in a transverse direction, at 26·5 cm. and

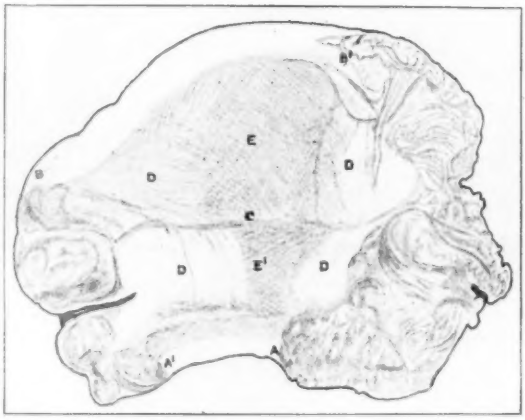

FIG. 5—Diagrammatic view of a transverse section of the tusk shown in fig. 3.

54·5 cm. from the pointed end. The diagrammatic view (fig. 4) of the upper surface of section B shows that the outside portion of the tusk from (*A*) to (*A'*) and (*B*) to (*B'*) is composed of a layer of cement showing a well-marked lamination. In the centre is a thin, darkish area (*C*) which marks the position of the pulp chamber. Between this area and the layer (*B*) to (*B'*) there is ivory, which in portion (*E*) presents the typical criss-cross appearance, while the portions marked (*D*) are denser in character. The tissues intervening between (*C*) and (*A*) to (*A'*) is also ivory, but only a small portion (*E'*) shows the typical appearance, the portions (*D D*) for the most part being much denser in character. In the area (*F*) the tissue is composed of secondary dentine. The area (*G*) is formed of ivory, bounded by areas (*H H*) of secondary dentine. An examination of the upper surface of the section C (fig. 4) discloses a similar condition to that seen in section B.

The relation of the tissues, as shown in the sections, indicates that the injury received, probably in a downward direction, split the tusk, depressing the upper fragment and causing an outward bulging at the sides, with considerable disorganization of the tissues in these positions.

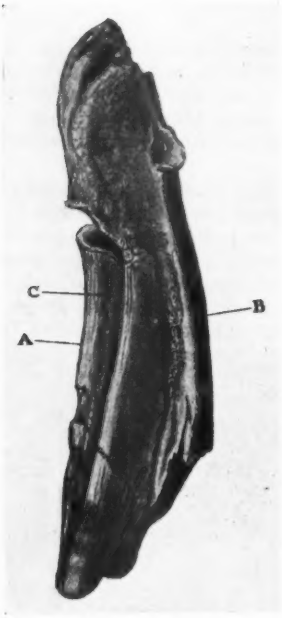

FIG. 6.

FIG. 7.

FIG. 6.—A tusk which has been split in a longitudinal direction. Roy. Col. Surg. (Odonto. Series), G122.3.

FIG. 7.—The tusk shown in fig. 6: view of the opposite side.

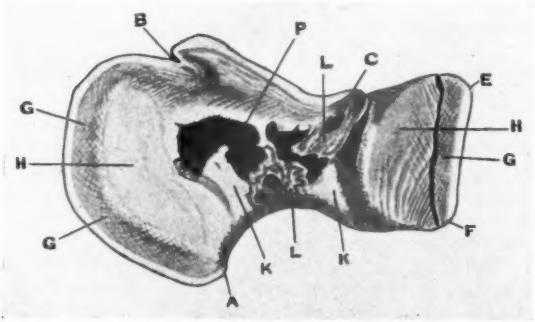

FIG. 8.—Diagrammatic view of transverse section of the tusk shown in figs. 6 and 7.

Figures 6, 7 and 8, are from the *Dental Record*.

The specimen (figs. 6 and 7) is the tusk of an African elephant which has been split in a longitudinal direction into two portions (*A*) and (*B*). The fragments have been joined together by the formation of fresh tissue. The portion (*A*) is regular in shape except at one part (*C*) where it appears to be folded and presents the appearance of a double tusk. This portion (*A*) is separated from the base of the tooth by a gap which measures at the circumference 30 mm., and gradually diminishes in width towards the centre of the tooth; the gap communicates with the pulp cavity. The portion (*B*), averaging in width 75 mm., is regular in formation and at its junction with the base presents an opening oval in outline. On one side (fig. 6) the two portions are separated by a narrow groove which, for the greater part of its length, is traversed by a narrow slit; on the opposite (fig. 7) there is a wide shallow depression between the two portions, and this is pierced by two irregular openings which communicate with the pulp cavity. The extent and nature of the injury can be examined best in the transverse section (fig. 8) made through the tusk at 2 cm. beyond the projecting end. Starting from the left-hand lower side at (*A*) there is a layer of regularly formed cement which is continued around the tooth to (*B*), where

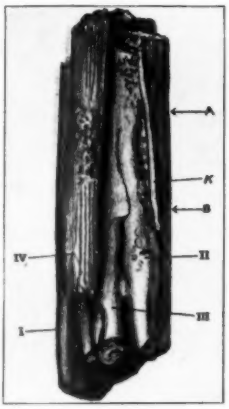

FIG. 9.—Portion of a tusk which has been injured. Roy. Col. Surg. (Odonto. Series), G122.4.

it is tucked inwards, and then continued to the position (*C*). Beyond this point, the layer of regularly formed cement is wanting, until the point (*E*) is reached, and from here to (*F*) it is again present and forms a well-defined layer. From (*F*) to (*A*) the defined layer of cement is absent. The inner aspect of this layer of cement is bordered by dentine which, for a distance of on an average 20 mm., shows the typical pattern of normal ivory (*G*). Beyond this is ivory of a dense character showing little or no pattern (*H*). The two portions of the tusk have been joined together by secondary dentine (*L*), in which are enclosed splinters of the original dentine (*K*). The position of the pulp cavity is shown by the letter (*P*).

The tusk (fig. 9) shows a stage in the formation of subsidiary tusks. The free end of the tusk has been broken in a transverse direction subsequent to injury. For convenience of description the tusk may be divided into five portions, I, II, III, IV, and K, fused together at the upper end, but separated at the lower end, the part K for the greater part of its length forming a distinct tusklet. The relations of these portions, the one to the other, are shown in the diagrammatic views of

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sections made 12·5 cm. and 20 cm. from the broken end. The arrangement of the tissues at the position (*A*) is seen in the drawing (fig. 10). Starting from the top left-hand angle (*A*) and moving to the right there is a regular layer of cement until (*B*) is reached; here for 2 cm. the tissue is of an indifferent type; then follows, at (*C*), a layer of regular cement, which continues until the upturn of the indentation at (*C'*), where there is a short break. A regular layer of cement forms the roof of the indentation: at (*D'*) there is a sharp line of demarcation, and from here

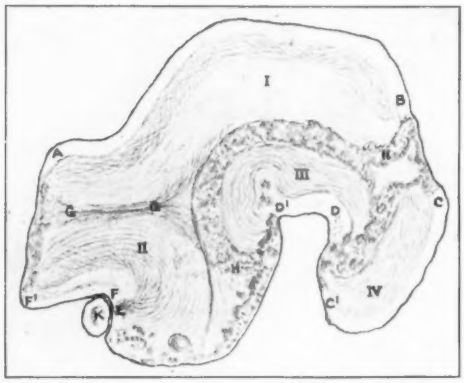

FIG. 10.—Section, at position A, of the tusk shown in fig. 9.

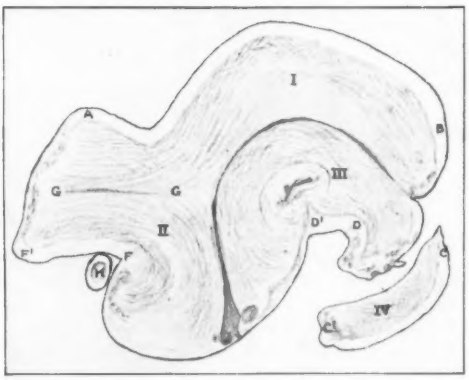

FIG. 11.—Section, at position B, of the tusk shown in fig. 9.

the outer layer as far as (*E*) is tissue of an indifferent type; from (*F*) to (*F'*) there is a layer of cement, and from (*F'*) to (*A*) indifferent tissue. On the inner aspect of the cement layers there are areas of well-formed ivory, which, for convenience of description, are numbered I, II, III, IV. These areas are separated the one from the other. Between the areas I and II there is a dark layer of tissue (*G G*); the area I is separated from the area III, and area III is divided off from II and IV by a wide layer of secondary dentine, H. K is a section through a detached tusklet. The

arrangement of the tissues at the position (*B*, fig. 9) are shown in the diagram (fig. 11). It will be noticed that the tusk is, in this part, composed mainly of well-formed ivory, and there is very little secondary dentine. The areas I, II, III and IV, somewhat ill-defined in section *A*, are now well defined. Area IV is separated completely from the main part of the tusk; there is a definite fissure between areas I and II, and area III is almost separated from the rest of the tusk. As the terminal part of the tusk is approached, these four areas become completely isolated, the one from the other.

The specimen (figs. 12 and 13) consists of the terminal portion of a tusk which has been injured. The injury caused a distortion of the developing tooth and

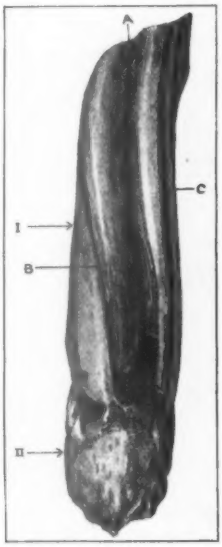

FIG. 12.

FIG. 13.

FIG. 12.—Portion of a tusk which has been injured. Roy. Col. Surg. (Odonto. Series), G 122.5.
FIG. 13.—Another view of the tusk shown in fig. 12.

complete arrest of growth. The tusk has been broken in a transverse direction at a subsequent date. The tusk has a pronounced tendency to a spiral curve and is deeply indented, the depth of the indentation becoming greater as the terminal part of the tusk is approached. On either side of this deep indentation, and following a somewhat similar course, is a shallow groove. The terminal part of the tusk is formed of a cone-shaped mass of tissue. This mass of tissue is firmly united for about half its circumference with the main part of the tusk; in the other half it is separated by irregular gaps. The tusk has been divided at the positions I and II. The relations of the tissues at the position I are shown in the diagram (fig. 14). The greater portion of the tusk is made up of well-formed ivory (I), with the laminæ conforming to the contour of the tusk; this formation of ivory diminishes towards

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the terminal portion of the tusk, and is united with this part by two tongues of cement. On the outer side of the ivory, except in the position (*G G*) is a cover of cement (*D*). The positions of the indentations are shown by the letters *A B C*. It will be noticed that the indentation (*B*) is continued into the body of the tusk, and that it is surrounded by a thin layer of cement. The lesser portion of the tusk is composed of secondary dentine, in which are embedded fragments of ivory (*F*).

The arrangement of the tissues in the terminal part of the tusk is shown in the diagram (fig. 15). The main part is formed of fragments of ivory (*F*) embedded in a secondary dentine, which contains many seed-like masses of tissue. Surrounding this is a layer of tissue (*D*), which, to the naked eye, has the appearance of osteo-dentine; outside this is a coating of cement.

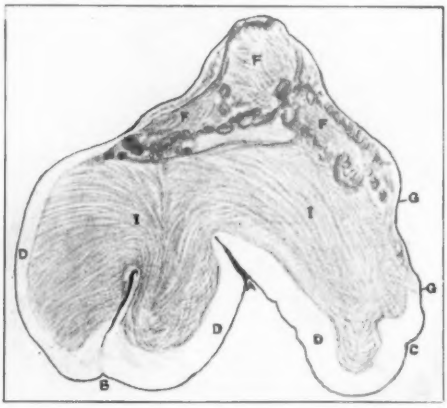

FIG. 14.—Diagrammatic view of a section made through the tusk, fig. 12, at the position I.

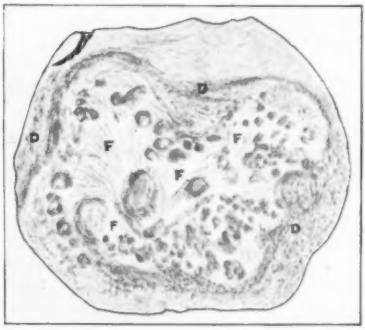

FIG. 15.—Diagrammatic view of a section made through the tusk, fig. 12, at the position II.

Section of Urology.

President—Mr. Frank Kidd, M.Ch., F.R.C.S.

[*March* 22, 1928.]

DISCUSSION ON THE TREATMENT OF URETHRAL STRICTURE AND FISTULÆ BY EXCISION.

Mr. Frank Kidd (President).

Abstract.—For hard tunnel strictures and in cases of perineal fistula we should be bolder to adopt the principle of excision rather than that of mere external urethrotomy.

A preliminary suprapubic cystotomy is advised, and also the avoidance, as far as possible, of the indwelling catheter. All fistulæ should be excised completely, not merely opened, scraped and drained.

The operator should not be in too great a hurry to pass sounds of too large a calibre after the operation, as in many cases there is little tendency to re-formation of the stricture.

The history of the operation of excision of urethral stricture, with a list of references, is summarized by MacGowan in the *Journal of Urology*, 1923, vol. x, No. 6. He points out that there are two types of the operation, one practised by Albarran and his pupils, a partial excision, where a strip of mucous membrane is left on the roof of the canal; the other advocated by König, and elaborated by MacGowan, of Los Angeles, and Russell, of Melbourne, in which a large portion of the urethral canal, including the roof, is cut away, the cut ends of the urethra being mobilized and brought together again by suture. The latter, or complete operation, is based on the now accepted teachings that there is no risk to the blood-supply of the urethra after cutting out a portion of the canal, and no risk of endangering the power of erection. It is advocated for this operation that there is little tendency to recurrence of stricture afterwards, so that it largely does away with a long course of after-treatment by means of bougies and the Kollmann dilator. The partial operation is described in Albarran's text-book, and also the method of complete excision in two stages elaborated by Pasteau and Iselin.

I will now demonstrate, with the aid of a series of diagrams, the chief steps of the complete operation as put forward by its chief protagonists, Russell and MacGowan.

Russell does not open the bladder, but is content to expose the deep urethra behind the stricture, according to Young's method for perineal prostatectomy.

MacGowan advocates a preliminary suprapubic cystotomy for two weeks before the actual operation. He also advocates injecting the urethra with methylene-blue before the operation, which stains the urethra and helps to map out the track of the stricture.

The steps of the operation are as follows:—

(1) The incision in the perineum is an inverted "Y." (Fig. 1).

(2) The musculature of the perineum is exposed and defined forwards as far as the testicles, or even further if necessary (the scrotum in that case being split), and backwards to expose the central perineal tendon. (Fig. 2).

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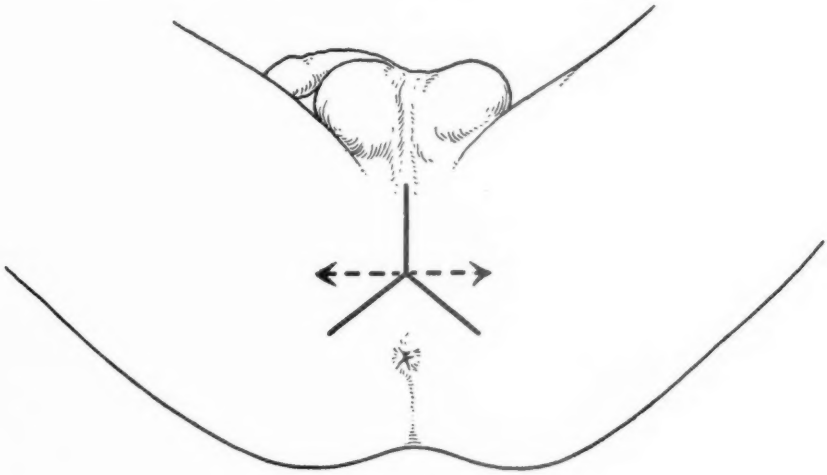


FIG. 1.

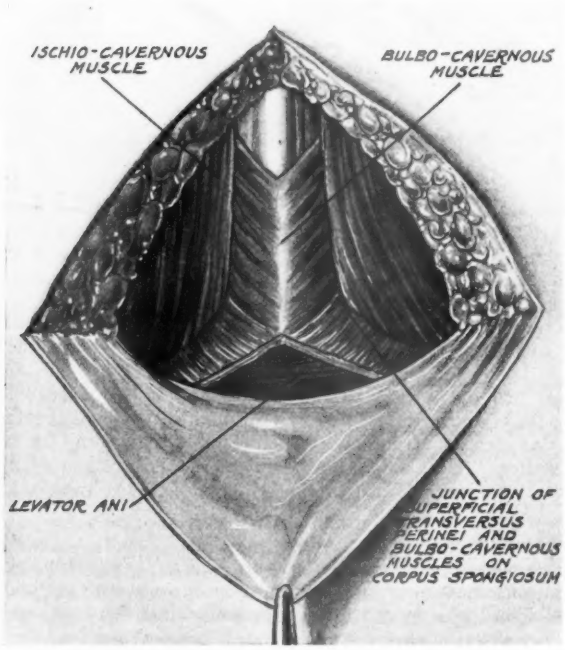
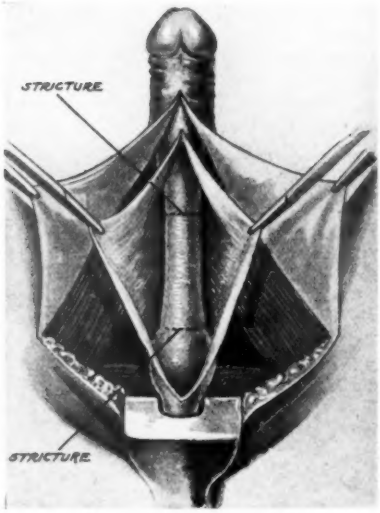
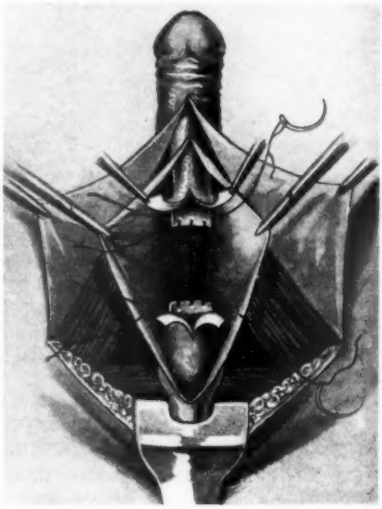

ISCHIO-CAVERNOUS MUSCLE
BULBO-CAVERNOUS MUSCLE
LEVATOR ANI
JUNCTION OF SUPERFICIAL TRANSVERSUS PERINEI AND BULBO-CAVERNOUS MUSCLES ON CORPUS SPONGIOSUM

FIG. 2.

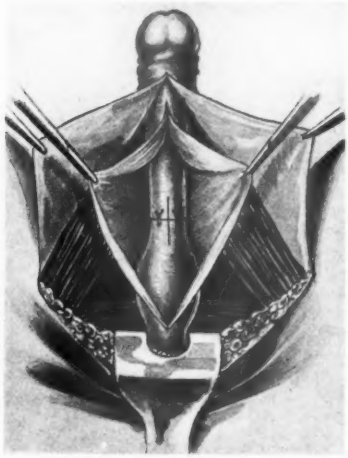


A.—Step 3.



B—Step 9.

FIG. 3.



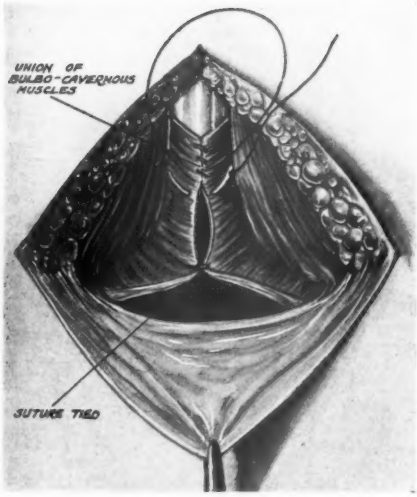


FIG. 4.
(MacGowan) Step 9 complete.

(3) The central tendon of the perineum is put on stretch by means of Young's retractors and divided close to the bulb. The central tendon of the bulbo-cavernosus muscle is split from before backwards, and the muscle pushed aside from the corpus spongiosum surrounding the urethra, and if necessary the transverse perineal muscles are cut and all blood-vessels ligatured. Russell also cuts the recto-urethralis muscle to expose the membranous urethra. (Fig. 3, step 3).

(4) A Wheelhouse staff is passed as far as the stricture, and the urethra is opened freely in front of this and held aside by sutures.

(5) A retrograde bougie having been passed from the bladder, the deep urethra is opened on this behind the stricture and the urethra held aside by sutures. In Russell's operation the deep urethra, having been exposed as in perineal prostatectomy, is opened behind the stricture.

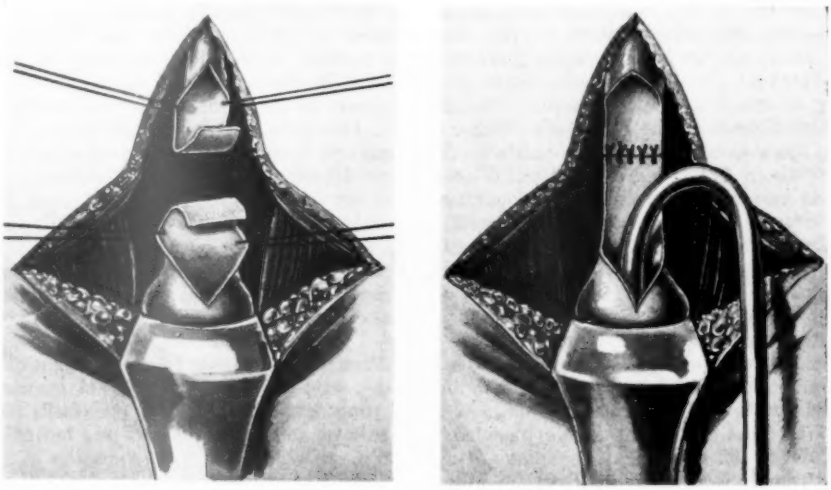


FIG. 5.
(Russell) Step 9.

(6) The tunnel of the stricture is then followed up and slit up on its lower face from the healthy urethra behind to the healthy urethra in front.

(7) The strictured portion of the urethra is now cut away altogether, as compared with the partial operation where a strip of mucous membrane is left on the roof of the urethra. MacGowan has removed as much as three inches of the urethra and has obtained perfect healing.

(8) Russell now mobilizes the urethral stumps, makes flat ribbons of the urethra in front and behind, stitches them together to form a new roof for the urethra, then fixes a perineal catheter into the bladder and leaves the floor of the urethra and the front of the wound open. (Fig. 5).

MacGowan passes a catheter from the meatus and stitches it into the deep urethra, splits the mucous membrane of the two portions of the urethra into three ribbons and stitches these up completely around the catheter. He then closes the perineal muscles around the urethra and catheter, leaving a small space for drainage. He leaves the suprapubic tube in the bladder for fourteen days. (Figs. 3 and 4).

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Russell and MacGowan both remove the urethral catheter in ten days, and at the end of two or three weeks pass very gently once a week a sound of only moderate calibre, from 18 to 20 on the French scale. No attempt is made to dilate the stricture for fear of tearing the junction. They state that healing is rapid and that there is no tendency to re-formation of stricture. MacGowan informs me by letter that one of the cases reported in his paper, on page 447, has had no indications of retraction of the stricture since February, 1921. I think this gives a fair description in as short a form as possible of the operation as published.

I have always had a feeling that there might be certain objections to the operation. I would like to know if there is any risk of incontinence from exposing the deep urethra so far back and cutting the compressor urethræ muscle, such incontinence as may occur after operations for perineal prostatectomy. One also wonders whether the advantages of the operation have been exaggerated. Does it make the passage of bougies after the operation less necessary? Again, some of the authors who have written on this subject seem to be a little hazy on the exact indications for the operation. From their papers, I imagine that they do not advocate it in cases in which simple dilatation can be carried out and proves effective, or in cases in which internal urethrotomy can be performed, followed by high dilatations with the Kollman's dilator. I have been able to obtain a permanent cure in the majority of cases so treated. For instance, I saw only this week a patient on whom I operated in 1915 and dilated up to 45 with the Kollman's dilator. He has had no trouble since the operation, and on examining him I found that his stricture had completely disappeared. I could bring forward a series of cases illustrating successful treatment along these lines.

Personally, I have only employed excision of stricture for certain types of cases, namely: (1) Where a hard tunnel stricture can be felt in the perineum from outside which refuses to respond to dilatation; (2) where there are perineal fistulæ through which pus and urine escape.

When in charge of the Genito-Urinary Department of the London Hospital, I was accustomed to operate on a number of patients, mostly of the sailor class, with old strictures and perineal fistulæ. At first I tried external urethrotomy, as described in the text-books, but I soon decided that this operation was useless and harmful. I developed gradually a technique of my own for these cases. They appeared to do extremely well treated along the following lines, which embody the principle, not only of excision of stricture, but also of the complete excision of a septic wound followed by partial primary suture, a principle which proved so successful during the war in dealing with gun-shot wounds.

I invariably carried out a suprapubic cystotomy at the commencement of the operation. This enabled me to pass a retrograde bougie, which was held in place by an assistant while the patient was put into the lithotomy position. I then cut down on the perineum through an inverted "Y" incision, cutting out freely all fistulas and all fibrous tissue connected with them. *Success can only be obtained by cutting out all tracks and their surrounding thick fibrous walls freely and boldly, paying no regard to the superficial tissues and only taking care not to cut the compressor urethræ muscle.* Complete excision of septic tracks is far better than attempts to open up tracks, scrape and drain them. I then defined as clearly as possible the perineal muscles and, after dividing the central perineal tendon but sparing the recto-urethralis muscles, I divided the bulbocavernosus tendon, so exposing the corpus spongiosum surrounding the urethra in front of the stricture, which I defined carefully, and then opened the urethra on a Wheelhouse staff. I then found that my retrograde sound passed from the bladder made it unnecessary, in the majority of cases, to cut the posterior group of perineal muscles. The stricture

always seemed to lie in front of the triangular ligament, and one could cut out the stricture from before backwards as far as the retrograde sound which bulged forwards to meet one at the anterior layer of the triangular ligament.

I then mobilized the distal portion of the urethra. It always seemed a fairly simple matter to stitch the superior wall of the anterior urethra after mobilizing it freely to the superior wall of the membranous urethra without mobilizing the membranous urethra at all. I found the best way to bring the two ends of the urethra together without the risk of the stitches coming out was to use a double cross-stitch, which should include a good bite of the tissues outside the urethra. Having secured the junction of the roof of the urethra, I worried very little about the lateral walls or floor; I simply reconstructed these around a sound passed from the meatus, stitching the perineal muscle together again, rather than paying much regard to the mucous membrane. I then removed the sound, stitched up the superficial tissues partially around a gauze pack, and left the suprapubic tube in the bladder for a fortnight. About a week or ten days afterwards I passed a metal bougie of moderate calibre which generally dropped quite easily into the bladder. At the end of a fortnight I removed the suprapubic tube and very shortly the patient would begin to pass water, sometimes entirely through the urethra; in some cases there would be a perineal leak for a short time, but three or four weeks afterwards the wounds had usually healed up completely, the fistulæ had been cured and the patient was able to pass water through his healed urethra without difficulty. Sounds were passed for a time afterwards. The patients, being mainly seafaring men, were usually lost sight of.

To illustrate this operation I will quote the following case operated upon recently:—

A man aged 67 years, first examined in 1927, stated that he had suffered for twenty years with a venereal stricture, attended by imperative, painful and very frequent micturition with a very poor stream. Some six months previously perineal abscesses developed which burst and left him with several fistulæ through which pus and urine escaped, so that his condition was one of extreme misery. The urine was alkaline and full of foul pus. A stricture could be seen at the back of the bulbous urethra. The prostate felt tough and fibrous. The blood-urea was within normal limits.

An operation was performed under spinal anæsthesia, preliminary suprapubic cystotomy followed by a perineal exposure of the stricture defined by a retrograde sound from behind and a Wheelhouse staff from in front. The central perineal tendon was cut but not the recto-urethralis muscle. The stricture having been clearly defined it was excised together with a large mass of fibro-fatty tissue containing several fistulous tracks. Two upward projecting lappets of fibrous prostatic tissue were then excised with the Young punch. The roof of the urethra was reconstructed, the perineal muscles partially stitched up, leaving a large cavity for drainage which was packed with gauze. A Pezzer tube was inserted into the bladder and the suprapubic wound stitched up around it. This was removed on the fifteenth day. By the twenty-first day the perineal wound had soundly healed but the patient had passed no urine naturally. On this day gas was administered and a metal bougie of moderate calibre passed into the bladder without the slightest hitch. The patient passed all his water naturally by the twenty-sixth day, and thenceforth recovery was uneventful save for a mild attack of pleurisy.

He left the Home and presented himself for examination eight weeks later. He could hold his water for four hours and pass it freely. The urine was acid but still contained a good deal of pus. Metal bougies dropped into the bladder without the slightest hitch.

The patient was instructed to wash out his own bladder daily by Janet's method. A fortnight later the urine was clear of pus, the frequency normal, and there appeared to be no tendency to any re-formation of the stricture as viewed by the urethroscope.

I feel then that excision of stricture, partial or complete, should be employed more extensively instead of external urethrotomy, which is a hopeless operation for

cases of perineal fistula with stricture, combined with complete excision of the fistulous tracks; also for cases of tunnel stricture in the bulbous urethra, for cases of tunnel stricture of persistent type after ruptured urethra, and also I am not at all sure whether we should not be wise to try the excision of tunnel strictures in the penile portion of the urethra. These strictures usually prove very resistant to internal urethrotomy and dilatation. I think it possible that better results might be obtained by carrying out suprapubic cystotomy and boldly excising such strictures, mobilizing and bringing the cut ends of the urethra together. Unless suprapubic cystotomy is carried out there is a risk of leaving a penile fistula. The penile urethra heals slowly and should not be subjected to the strain of the stream of urine for at least fourteen to seventeen days.

Heitz Boyer has advocated immediate extensive and complete excision with suture of the pulped ends of the urethra when ruptured by violence, using suprapubic drainage, and being careful to omit the indwelling catheter.

In an article on ruptured urethra (*Rapport de la Société Internationale d'Urologie*, 1921) I have advocated suprapubic drainage in all cases of ruptured urethra, followed by perineal exposure, identification of the proximal end by means of a retrograde bougie, followed by suture of the roof only of the cut ends and leaving the perineal wound freely open without the indwelling catheter. Banks (*Brit. Journ. Surg.*, October, 1927) has described an ingenious method for dealing with this emergency, by retrograde catheterization through a suprapubic opening of the bladder without opening the perineal at all and without suture. The method of retrograde dilatation of stricture is recalled by this operation and is one of which we should like to hear further from any who have had special experience of it.

I would like to take this opportunity of bringing forward a device which I have employed for some time, and which I have found helpful in dealing with strictures of the urethra. Dr. Lennox Wainwright called my attention to the fact that it was possible to relieve cases of Dupuytren's contracture of the hand by means of regular doses of thyroid extract, and I found from personal observation that this was true. I have therefore been accustomed for some time in all my cases of stricture to give a grain of thyroid extract by the mouth every night for many weeks at a time, and I have gained the impression that this most distinctly softens the fibrous tissue of a stricture and renders it more amenable to dilatation.

Mr. E. T. C. Milligan.

For a clear conception of the pathology and treatment of stricture of the urethra it should be appreciated that the inflammatory process responsible for the condition is almost entirely confined to the roof of the canal.

The urethral glands and depressions infected in gonorrhœa are for the greater part situated in the roof of the anterior urethra, and, following their infection, inflammatory nodules are readily seen in the roof of the air-distended urethra, and occasionally at the sides, but very rarely in the floor (fig. 1). In most cases these disappear after a time under various forms of treatment, but it occasionally happens that fibrous tissue formation leading to stricture subsequently follows. I have observed this change on several occasions. First a "signet ring," then a "lunule," or "crescent" of fibrous tissue appears, which involves usually the roof, but sometimes either or both sides and roof (figs. 2, 3, 4). It is quite rare to see fibrous tissue first appear in the floor of the urethra The stricture becomes whiter in appearance towards its central lumen, indeed, with air-distension, at the extreme central sharp free edge it is almost translucent and appears entirely avascular. This appearance

FIG. 1.

FIG. 2.

FIG. 1.—Inflammatory nodules in roof of urethra. Air distension.
FIG. 2.—Early stricture of urethra. "Crescent" in roof and sides. Air distension.

FIG. 3.—"Crescents," "lunules," or early strictures, in sides and roof. Air distension.

FIG. 4.—Stricture, replacing nodule on roof. Air distension. Signet ring type.

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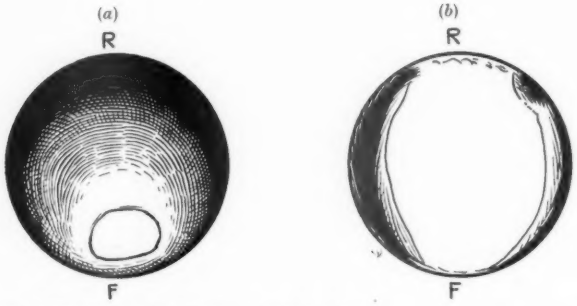
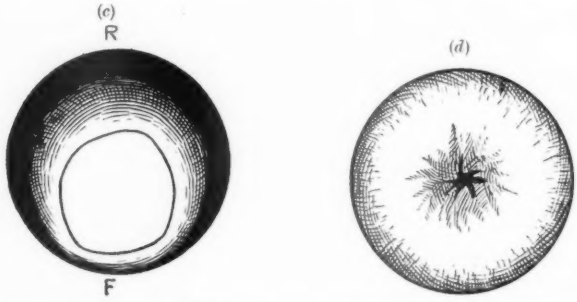
distinguishes it from the normal urethral folds. At its peripheral margin (although unfortunately it it so rare to see this stage and confirm this point in the dead subject) it appears to all tests to be confined to the mucous membrane and does not penetrate deeply into the subjacent layers. It still swings loosely with the mucous membrane.

This stage is readily detectable with the urethroscope. No case of gonorrhœa properly controlled should be allowed to pass further than this stage. It is easily and successfully treated by dilatation with sound and mechanical dilators. I will refer to it subsequently as Type I.



(*a*) Stricture of urethra before dilatation. Air distension.
(*b*) Appearance immediately after dilatation ; roof ruptured sides still present. Air distension.



(*c*) Appearance of stricture (re-formed) one week after dilatation. Air distension.
(*d*) Normal urethra. Not distended.

FIG. 5.

Later, however, in more advanced neglected cases the appearance and character of the stricture entirely alters. It loses its pearly-white colour and translucent edges, it becomes vascularized and appears light pinkish-white in colour. The free central sharp edge becomes more rounded, thick and irregular, and it is then less movable and even rigidly fixed on the subjacent tissue, indicating deeper permeation. Not only are roof and sides of urethra now involved, but also floor, though not usually to the same extent as roof and sides, so that the opening or lumen on air distension is usually eccentric towards the floor. Type II (fig. 5*a*).

Two factors are important and perhaps responsible for stricture formation.

The first is *mechanical. The normal position of the urethra is closed.* The mucous membrane falls loosely together by its own elasticity and its walls lie directly in contact (fig. 5*d*). The passage of urine at varying pressures, according to the power of the bladder or to obstruction to this passage, separates the walls, but rarely to their fullest extent. This can easily be demonstrated by pinching the urethra at the glans while urine is being passed, when the urethra will be felt to dilate further, even to its maximum. When micturition ceases, the urethra returns to its normal collapsed and closed position. Now it is an easy calculation to multiply the time it takes to empty the bladder by micturition and the number of times micturition takes place in a day, to appreciate that *for all practical purposes the walls of the urethra are in the closed position most of the day, and in most cases all the night.*

The other important factor is that the inflammatory products, outpoured plasma and cells, not only set a tissue in its normal, habitual position, but make it inelastic, *so that its function and property of elasticity is abolished.* In the case of the urethra, this function is dilatation and collapse. Later when fibroblasts appear and fibrous tissue is formed, elasticity disappears entirely and the urethra is firmly "set" in its normal position or a compromised position between the normally closed and very occasionally dilated position. Normal force of micturition would have little dilating effect on fibrous tissue.

If the urethra were a tube held open permanently, like a hose-pipe, there would be no stricture, for the inflammatory products and subsequent fibrous tissue would "set" the tube in its habitual position, i.e., open. The same reasoning applies to the œsophagus, the intestines, bile ducts, rectum and anal canal. Anatomical differences, variation in contents and varying function—which, however, do not concern us now—determine the different behaviour of these other tubes to inflammatory, inelastic tissue. The amount of actual active contraction that fibrous tissue is capable of is perhaps over-rated.

With regard to *excision of stricture*—the mechanism of its action, the indications for its employment—and an explanation of its proved satisfactory results can best be assessed by a reference to the other known methods of treatment and the aforementioned conception of stricture.

The well-established methods of treatment of stricture of urethra are :—

(1) Instrumental dilatation by sounds and mechanical dilators.

(2) Cutting or burning through a urethroscope, or by specially designed "blind" instruments, viz., urethrotomes.

(3) Cutting the stricture by an open operation through the perineum, the so-called external urethrotomy.

(4) The removal of all fibrous tissue by excision, and restoration of continuity of the urethral canal by suture.

In Type I (and may I remark that I only use these types for clearness in this description, for clinically the two types merge into each other), in which the fibrous tissue has not penetrated deeply into the subjacent tissue, dilatation is satisfactory. It easily establishes full dilatation, and elasticity of the urethra returns.

Dilatation breaks the avascular curtain usually in the roof, and in most cases little or no bleeding results. Viewed immediately afterwards with the urethroscope, one finds a few fine smears of blood, the stricture entirely disappeared, and the urethra dilatable by air distension to the same extent as the adjacent normal.

In other cases of perhaps a more advanced stage, only the dorsal part has disappeared, leaving an inconspicuous small raw area of healthy tissue. Full dilatation can be obtained in a vertical direction, but at the sides a portion of the stricture, pearly-white in appearance, remains projecting as a ledge.

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If no dilatation is practised for one week, one finds that the stricture is almost re-established. The parts have fallen together, the force and volume of micturition are not sufficient to separate them, and the old condition of affairs is nearly re-established. (Fig. 5, *a*, *b*, *c*.)

This can be entirely prevented by measures that will keep the urethra permanently distended, as, for example, an indwelling catheter; indeed, the mild inflammatory products will then tend, by abolishing elasticity, to prevent collapse of the roof into the normal closed position so favourable to re-formation.

When one uses the urethroscope freely, one will find that most strictures to be dealt with will come into this type. If one waits till there is interference with micturition, more of the next type will be encountered, for the permanent lumen of a stricture is very small before symptoms arise.

It is in Type II, ranging from advanced stages of white fibrous stricture through the vascular well-organized varieties up to the stricture with fibrous tissue penetrating to the perineum, that the problem of selecting the most suitable type of operation is difficult.

In all strictures, except those with perineal fistulæ and fibrous induration, dilatation should be given a trial, success being gauged by ability to attain and maintain full dilatation as observed immediately afterwards, and subsequently by the urethroscope.

FIG. 6.

FIG. 7.

FIG. 6.—Site of incisions in internal urethrotomy. Air distension.
FIG. 7.—Internal urethrotomy incision spread out by indwelling catheter. (Diagrammatic.)

Failing success, internal urethrotomy should be next practised. Internal urethrotomy is successful because the cut is made in the roof of the urethra where the fibrous tissue arises, and is usually most marked (fig. 6). Its success depends on whether the limited depth of cut which is possible is sufficient to penetrate past the fibrous tissue to healthy tissue beyond. This is subsequently held open by indwelling catheter for several days after operation (fig 7). Mild inflammatory products setting it in this open position and stiffening it prevent the urethra from collapsing entirely till epithelization is complete.

If this cannot be done the stricture will relapse, and although the patient states that he is better, yet his comfort will depend on frequent and perhaps difficult instrumentation over an indefinite period. The force of the urinary stream is not sufficient to dilate the inelastic fibrous tissue remaining; the amount of dilatation becomes less and less till symptoms recur.

If the cut cannot be made entirely through and beyond fibrous tissue into healthy tissue in the roof, then at a subsequent operation the same depth of cut made more

lateral may penetrate in this locality through and beyond the fibrous tissue, which is here perhaps not so deep. Excision of stricture, however, is perhaps better treatment if relapses occur.

It is common knowledge that internal urethrotomy is a most satisfactory operation. It is easy of performance and comparatively little inconvenience is suffered by the patient. Convalescence is short and after-results in well selected cases are all that could be desired. It is not likely to be replaced by excision, as the routine operation for stricture. Furthermore, it is applicable to multiple or single strictures in the penile urethra.

Yet, till we are able to select and classify strictures by means of the urethroscope or other clinical methods from the point of view of depth of penetration of fibrous tissue, there will be a small number of cases for which internal urethrotomy is unsuited and which will inevitably relapse after this operation, cases which were more suited for excision of stricture in the first place.

External Urethrotomy.—It is easy to explain the cause of failure to cure in external urethrotomy, indeed even to pass a sound after operation. The unimportant

FIG. 8.

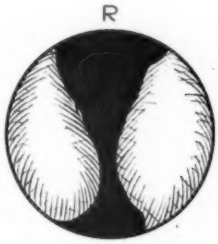

FIG. 9.

FIG. 8.—Open appearance of urethra after excision of stricture. Not distended.
FIG. 9.—Open appearance of urethra after excision of stricture. Not distended.

fibrous tissue in the floor of the urethra is incised in this procedure, whereas that in the roof, the important part, has been left untouched.

It is good practice in the operation of external urethrotomy to deal with the fibrous tissue in the exposed roof and at the sides by incision in these areas, much in the same way as they are dealt with in internal urethrotomy.

It is better, however, in my experience, to perform excision of stricture rather than external urethrotomy. It is an easy enough matter to excise a stricture of Type 1, and to join up the urethra again. It is essential to excise the stricture in the more advanced cases if relapse is to be avoided.

Excision of Urethra.—In performing this operation I follow Hamilton Russell in his essential points, removal of all fibrous tissue, suture of the spread-out "riband" urethra and formation of a wide urethral roof. My results are in full agreement with his published successful cases. Some light is thrown on the reasons for the success of the operation if one observes these cases afterwards with the urethroscope.

Instead of the urethra at rest being closed by its elastic walls, it is held permanently open on the roof where the line of union which replaces the various elastic coats of normal urethra is adherent to subjacent rigid tissue, outside the urethra, viz., the triangular ligament and subpubic tissues (figs. 8, 9). These structures are very well suited to hold the walls of the urethra apart. Furthermore,

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the open position does not interfere with the function of the urethra, for in front of and behind the area of excision the normal condition of affairs exists and the urethral walls close at rest.

Moreover, this condition of affairs lends support to the special technique of the Hamilton Russell operation which insists on a "riband" spread-out urethra being sewn together in the roof. It also stresses the importance of the tension sutures which in his second publication he suggests should be put through the outer coats of the urethra. I, wisely or not, included extra-urethral tissue in these tension sutures in the hope of encouraging adhesion of the newly formed urethral junction to subjacent tissue.

Excision abolishes all stricture tissue and local diseased follicles which the other methods fail to do.

It holds the urethral walls apart immediately after operation—a critical and important time—till they are set in this position by outpoured lymph and subsequent scar-tissue, resembling somewhat in this the action of an indwelling catheter in internal urethrotomy. The subsequent scar tissue has the effect of keeping the walls apart, as it were pinning the urethra to surrounding firm structures. Further contraction of scar-tissue, if it does occur, would be beneficial, as it would hold the walls more securely open.

Relapses (and cases do relapse after from one to four years) are due to the urethra becoming detached and more movable again, conforming, in effect, to its original normal closed position. For this Hamilton Russell recommends passing a full-sized sound at yearly intervals.

The main criticism of the operation is, that with the use of the urethroscope it should never be required; other much less severe measures, viz., dilatation and internal urethrotomy, sufficing.

In cases where I have excised from 1 in. to 2 in. of the urethra, great foreshortening of the penis has resulted. Some may regard this as objectionable. In cases of multiple strictures, I have excised the most extensive one in the perineum and dealt with those in the penile urethra by internal urethrotomy.

Indications for the Employment of Excision of Stricture.

Hamilton Russell writes—"in all cases not easily managed by dilatation." I would add to this "and by internal urethrotomy."

If one could estimate the depth in the urethra to which fibrous tissue had extended and one knew the depth to which the incision with the urethrotome would penetrate, then the selection of cases for suitable operation would be easier. Up to a point this can be guessed at from the appearance of the stricture as seen by the urethroscope.

All cases of perineal fistulæ with fibrous induration associated with stricture are fit cases for excision. It is unwise to try other methods. I have had satisfactory results in five cases of this nature.

For success all fibrous tissue wherever encountered should be relentlessly excised, and all tracks followed even to inguinal regions and lower abdomen. Healthy tissue should bound all wounds. All fibrous tissue must be cut from the urethra.

As long as some shred of urethra can be sewn together in the roof without tension, then the perineal floor is not important, for where much skin has been sacrificed closure of the floor can be effected later after healing of the roof of the urethra has occurred, a condition resembling perineal hypospadias being brought about. I have been much interested to observe the healing of the joined "riband" urethra in such an open wound. Most of my cases have been drawn from patients

who, by reason of their occupation, nationality or sensibilities, are not likely to subject themselves to intermittent dilatation.

Lastly, I would like this opportunity to congratulate Mr. Hamilton Russell (a man beloved by all his pupils, himself a worthy pupil of Lord Lister) for giving to the surgical world a technique in the excision of stricture generally applicable, but without which some cases of stricture would be condemned to permanent suprapubic drainage, the urethral condition being considered incurable.

With his method, I have rescued at least two cases thus condemned, after the trial (without success) of all other known methods to effect a cure.

Mr. R. Ogier Ward

said he did not make use of the retractor, for, without that, it was not difficult to come down on the urethra in the way the President had described. Having exposed the corpus spongiosum, one could easily feel the stricture. He then worked in a direction away from the diseased part, going well forward to dislodge the whole corpus spongiosum from its bed. With a little dissection, using curved Mayo's scissors, one could pass one's finger round behind it. Thus one cleared the whole circumference of the urethra in front of the stricture.

As to defining it behind the stricture, he had never yet opened the bladder in these operations, but he had not dealt with any form of fistula, such as the President had. If one was going to deal with fistula, the bladder should be opened first. He had not opened the bladder in the cases he had dealt with, as they were cases of straightforward single stricture of the bulb, with one or two exceptions. If a steel bougie from the bladder was in position, the urethra behind the stricture would, of course, be brought into view; otherwise, careful dissection had to be done to expose it. Usually the speaker had found this dilated part, opened it, and excised the strictured part. When he had difficulty, the whole corpus spongiosum being mobile in front, he cut it across just anteriorly to the stricture, then cut away the thickened part and thus found the healthy urethra behind it. Then he began to suture the torn ends together.

Hamilton Russell had said that the catheter passed into the bladder must not lie on the suture line. That was all right in the condition depicted in the President's drawing; but when the stricture was close to the triangular ligament it was difficult to get the catheter to lie so that it was not in contact with the suture line. Still, his cases had apparently healed all right. If all strictures happened to be half an inch in front of the triangular ligament, the operation would be an easy one. Sometimes, however, the stricture was very close to the triangular ligament, and then the operation was difficult. In his last case there was no healthy urethra in front of the triangular ligament, indeed the stricture spread into the membranous urethra, and he had considerable difficulty in finding it there—he had to dilate it up with steels before he could suture the parts. Also there was trouble about healing afterwards. It was healed now, however, and instruments could be passed.

It was important not to suture only mucous membrane; the whole thickness of the corpus spongiosum should be included in the sutures. If that was not done the sutures were apt to cut out.

Dilatation was a very good way of treating strictures; so was internal urethrotomy; but if one got a case such as he would sketch (drawing), the guide could not be got through, and then was left with the alternatives of excision of the stricture or external urethrotomy. The latter he did not regard as a very useful procedure. It was the old operation of emergency, but he did not think anyone would employ it

now for retention of urine; they would do cystotomy. In such cases as he had depicted, in which an instrument could not be got through, if was better, if there was no emergency, to make a careful dissection and excise the strictured part.

Mr. Fowler Ward

said that his first case in genito-urinary surgery—ten years ago—had been in a man who had several sinuses in the perineum and groin. He had taken the "bull by the horns," cutting open and excising all the strictures. The patient had recovered. His procedure was now as follows: If the urethroscope showed a diaphragm stricture, it was likely that the condition would respond to dilatation, and if three or four dilatations were successful there would probably be no further trouble. If, however, it did not respond to these efforts, there was no alternative but external urethrotomy. Internal urethrotomy was working in the dark, and occasionally left ghastly results. The arteries came up under the pubic arch, and if an important artery was cut the only way to stop the bleeding was to tie it as in Pearce Gould's operation for total extirpation of the penis. If a patient had various bad strictures, one cut down on to the urethra, and if he was able to pass a guide of any sort it helped him in the operation greatly. If he could not pass a filiform bougie into the bladder, he opened the bladder and did a retrograde catheterization. He preferred a metal catheter; it came through the bladder and indicated where the urethra should be. He cut through the stricture, and then did what seemed best for the particular patient. A silver catheter could be left in for a week, without causing sepsis. A soft rubber or gum-elastic catheter was pitted when it had been in a week. But there were cases in which the silver catheter could not be passed, as there was a large quantity of fibrous tissue all round. Then the only course was complete excision, especially if sinuses led from the pubes or the perineum. The bleeding was not very severe. In only two of his cases had there been serious bleeding, and he believed it was bladder bleeding in both, not from what was cut.

He remembered the case of a man who had had extravasation of urine after a severe injury. Two or three inches of urethra in that case had "gone to the bad," but after a time the wound had healed. There had, however, been adhesion to the perineum, and every time an attempt at dilatation was made the perineum had given way. He (the speaker) had then performed a plastic operation, turning a flap over from one side to the other. Healing had been very satisfactory. Afterwards, looking down the urethroscope, he had seen a quantity of hair. The patient remained well for four years, after which time there had been difficulty in passing a bougie. He (the speaker) then thought he would remove the hairs seen after the previous operation. He again performed external urethrotomy, and found that the hairs were very fine and very few; the urethroscope had magnified them so that they seemed likely to cause obstruction. They were destroyed with the cautery, and now the patient had a perfect urethra, formed of external skin. Whatever might be the microscopical structure of tissue inserted in the urethra, it acted very well. This patient was now quite healthy.

The President had mentioned strictures of the anterior urethra. Recently he (the speaker) had two cases of stricture involving the anterior; 3 in. were involved, i.e., extending to the penile-scrotal junction. One of the patients was a man aged 70, who, for some reason, had sudden retention, and nothing more than a filiform bougie could be passed. On his (the speaker's) instructions his house-surgeon had performed a suprapubic cystotomy. Later he made an artificial meatus in the perineum and could only pass a filiform bougie from there to the end

of the penis, therefore he (Mr. Ward) did a modified Duplay's operation. He took a longitudinal flap of skin and from it constructed the whole anterior urethra. Instead of finding the corpus spongiosum of a certain amount of thickness, he found only what was practically a thin fibrous tube. After the operation the man retained for some time a suprapubic and perineal fistula. The operation was only done eight weeks ago, and yesterday he was able to pass a No. 17 bougie from the anterior meatus into the bladder. So pleased was he with this result, that a week later when another man came with apparently the same condition he decided to treat him likewise. The history this patient gave was that he had been bitten there by a dog. He said that when he passed water it blew out between the penis and the scrotum. This man was only 55 years of age, and as the tissues were probably good he (the speaker) decided to operate. In five days, however, the whole thing sloughed. Hugh Young made the important suggestion that plastic operations should not be done on the anterior urethra unless there was drainage from the bladder or the perineal wound. In this case he did not take that precaution, and the whole penis sloughed in a few days. The patient, however, was pleased because he could pass his water, although through the wrong site.

An interesting question was as to why these strictures of the anterior urethra occurred. They were said to be congenital, but he was not convinced about that. In the first of the two patients spoken of the condition did not occur until shortly before 70 years of age, and the other was 55.

A few days ago a man came on whom he (the speaker) had done a meatotomy five years ago. In that operation he tied the mucous membrane of the glans to the skin—Hope's method, he believed—and he always passed a full-size Beniqué catheter through the meatus to test whether there was a good urethra. This man had a stricture of the meatus from the previous meatotomy, and the speaker thought it would be right to do a meatotomy again. He had a stricture extending 3 in. down the anterior urethra. Apparently one could get an ascending fibrosis of the penis, and the corpus spongiosum atrophied. In future cases he intended to open the bladder or make an artificial meatus in the perineum; he believed that would give a successful result.

Mr. H. W. S. Wright

said that while working in the Far East he had performed this operation for stricture many times, but had seldom seen the type of stricture common in England. As the patients came from great distances, and as most of them were poor, a visit to the hospital was a serious undertaking for them. Consequently only the more serious type of stricture came for treatment. A large proportion were traumatic; the rest were old gonorrhœal strictures which had been mishandled. In China retention was often treated by the passage of a long steel needle, which might or might not give relief; in any case the ultimate result was a false passage and great fibrosis. Many of these strictures were easily palpable from the perineum and were often half an inch long, and complicated by extravasation, fistula, or abscess formation. In such cases there was no question as to what type of operation should be done. The patients would not remain long enough for regular dilatation; often an instrument could not be passed, or if a urethrotome guide would pass the staff itself was too large to do so.

He began by treating them as he had been taught in England, namely by dilatation with filiform bougies, and later internal urethrotomy, but found that this procedure had several disadvantages. Sometimes it was impossible to pass a guide,

and patients would not come for post-operative dilatation; he had seen two cases of extravasation after the operation, even though the catheter had been retained.

In each case the stricture had been very long and fibrous, and he had used a high urethrotome knife, which, as well as cutting the stricture, had opened into the tissue above; he therefore abandoned this method for that type of case.

With regard to the technique of the operation itself. The incision and exposure described by Mr. Kidd had logically imposed itself upon him. He had had many anxious moments finding the posterior urethra, but had comforted himself with the same reasoning as Mr. Ogier Ward's, namely, that behind the stricture the urethra was always dilated. He cut down on a Wheelhouse's staff, defined the face of the stricture, and then sliced the urethra transversely till he came to the dilated urethra at its back. He regarded mobilization of the urethra as a very important part of the operation, and thought the method described by Mr. Ward was useful. After excision it was well worth taking much trouble to secure approximation of the edges all round a catheter passed into the bladder, because whenever there was a gap in the floor of the urethra there would the fibrous tissue be gathered together, as was shown in the illustration of a previous speaker; he had often proved this by urethroscopic examination. He had come to the definite conclusion that suprapubic drainage had no advantages over perineal drainage for the ordinary case. It was recommended by the French school, and he tried it in about ten cases and found its results no better, and sometimes worse than perineal drainage. Naturally he had used it on the worst cases. In one case, complicated by extravasation, he had done two external and three internal urethrotomies. This patient lived too far away to come for dilatation.

If he could do so without going behind the triangular ligament, he used a method of drainage suggested by Dr. Char, of the Pekin Union Medical College. A small incision was made half an inch behind the sutured urethra on to a catheter passed into the bladder, and through this incision he passed another catheter for drainage. This method gave as good results as suprapubic drainage.

From increasing experience he had come to believe that it was wise, very gently, to pass an instrument as early as the fifth day after operation. It was passed as far as the retained catheter and prevented adhesions between the floor and the roof of the urethra. Later, when the retained catheter had come out, a large sound could be easily dropped into the bladder.

In suturing the urethra, he thought with Dr. Kidd that it was important to use figure-of-eight sutures, which took in fairly large "bites" of the spongy tissues, rather than small sutures which only penetrated the mucous membrane.

In one of his first cases the patient had had partial incontinence at night, probably due to interference with the sphincter.

All his cases had not been complete cures. Four, all severe cases, had come back for internal urethrotomy. The worst cases were traumatic strictures complicated by extravasation. It must be remembered, in assessing these end results, that very few patients could attend for post-operative dilatation.

To sum up, there was no doubt that for severe tunnel strictures, excision was much better than internal urethrotomy. He had seen several cases, from one to three years afterwards, who were quite comfortable, and who had never had an instrument passed since they left hospital.

Mr. A. P. Bertwistle

said he had seen a number of cases operated on in Leeds. Some had been perfect cures, but in those in which the stricture had re-formed, the last condition was worse than the first. No doubt, in view of what had been said by Mr. Kidd, the excisions had not been wide enough and diseased tissue had been left behind.

Mr. Clifford Morson

wondered whether the strictures met with in Australia were worse than those in England; in China they were very bad. In his experience in recent years at St. Peter's Hospital, there had been marked improvement in the extent of stricture formation. When he was house surgeon, internal urethrotomy was a common operation, now it was very unusual.

One particular indication for excision was where the stricture was caused by trauma. He believed that in twenty years a discussion on the operative treatment of stricture would not have a practical aspect for the Section of Urology. Prevention was better than attempted cure, and those specializing in the treatment of venereal disease were making greater progress than those who called themselves urologists; before many years had passed it would be very uncommon for a man who had had gonorrhœa to have stricture of the urethra. Trauma was a different matter, and for that, excision would, from time to time, be necessary. But even in cases of trauma, if one followed out the technique to which Mr. Hamilton Bailey drew attention in *The British Journal of Surgery*, the late results of traumatic stricture should be extremely good. Many of the severe strictures of the urethra were produced, he suggested, by maltreatment by one or more medical men.

Mr. H. P. Winsbury White

said that several speakers had compared internal urethrotomy with external urethrotomy, as if those operations possessed equal merits, whereas those who practised both extensively considered the internal operation far superior. The worst strictures he had had to dilate were those after external urethrotomy. He had not found it necessary to perform the external operation once during the last five years. Occasionally he saw a stricture which would not admit a filiform bougie; but most surgeons who were accustomed to doing internal urethrotomy had discovered that there very few strictures which could not be negotiated with a filiform bougie, especially of the corkscrew type, by exercising patience and using the right kind of guide.

Strictures in the anterior urethra were not, as Mr. Fowler Ward appeared to think, unusual. Those who used the urethroscope frequently knew that these strictures were common, though they were not, as a rule, so far advanced as those in the bulb. But in the average case of stricture in the bulb, if one examined 2½ in. of urethra, posterior to the navicular fossa, there would be seen a number of early lunules which had a relationship to follicles. The stricture was really an extension of the inflammation at that spot. The reason why internal urethrotomy was so successful was because all the early strictures could be divided with the urethrotome. Follicles were opened up in addition to dividing the stricture. It was a striking illustration of what was frequently seen in patients who, ten or fifteen years previously, had had gonorrhœa. The case [illustrated on screen] was not definitely a stricture case; the patient had had a small gleet. A fairly large-sized instrument passed quite well into the urethra, but with the urethroscope one saw what was depicted; and the smallness of the orifice of the follicles was striking. Normally, the orifice of a follicle was a flat, shallow depression, and the result of long-standing infection was to make it elevated and constricted, so that the gland became a distended sac.

Another case [illustrated on screen] was one in which dilatation was not possible; it was dealt with by diathermy. He had seen two similar cases in twelve months. Air-distension gave a heart-shaped stricture [drawing shown], and the accompanying illustration [shown] depicted the same condition without air-distension. He had

been able to pass a large sound. There was a slight gleet; the patient had had gonorrhœa fifteen years before. A large sound had entered the bladder without obstruction. He (the speaker) used, in the diathermy treatment, Joly's posterior urethroscope, and divided the posterior limb. Underneath it he discovered the mouth of the follicle, and this he burned out. He had treated another case of the kind in the same way, and with equal success.

Mr. G. P. B. Huddy

said that the results of internal urethrotomy depended almost entirely on the patient attending hospital regularly for dilatation afterwards. In his experience less than half of these patients did so; consequently they relapsed.

How did Hamilton Russell's excision results compare with those of internal urethrotomy with regard to the frequency of subsequent dilatation?

Mr. Frank Kidd (President) in reply

said that he had learned a good deal from this useful discussion. Much more was to be made of excision in these cases than was generally realized. He agreed that internal urethrotomy was a very useful operation, but external urethrotomy was a useless operation, and should be discarded. At the London Hospital there were many patients of the sailor class who, on admission, were in a terrible condition owing to fistulæ. He always excised these fistulæ completely, as he realized he had got to do that in order to effect a cure. It was astonishing to find how well they did after excision. And his impression was that they did not need so much dilatation afterwards.

With regard to the urethrotome knife, he recently went to operate at a hospital where he had never operated before, and he did not take his own urethrotome with him. He was horrified by the knife in the urethrotome which was brought to him. But he had to use it, and it caused much bleeding. In his own instrument he used a very small knife; one did not need to cut deeply. It was best to make a small incision, and then to stretch with large bougies. The danger in internal urethrotomy was in the use of large knives, such as those usually provided by the instrument-makers. He had never had serious bleeding after internal urethrotomy, using small knives.

The principle of excision should be accepted both for perineal fistulæ and for difficult strictures. The keys to the operation brought out by the discussion appeared to be:—

(1) Division of the bulbocavernosus muscle in the middle line, permitting of blunt exposure of the corpus spongiosum.

(2) Blunt dislocation of the corpus spongiosum containing the urethra in front of the stricture.

(3) The resection of the stricture backwards from the urethra so freed and opened until the dilated urethra behind the stricture was opened up.

(4) Suture of the roof of the urethra.

It seemed to be an open question as to whether better results could be obtained by a preliminary suprapubic cystotomy with drainage, or by perineal drainage through the deep urethra.

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[*May* 24, 1928.]

Observations on the Radium Treatment of Vesical Carcinoma.

By A. Clifford Morson, O.B.E., F.R.C.S.

Abstract.—A review of the position of radium-therapy in 1913 and of the knowledge of this subject acquired by the year 1914.—The discoveries of the relationship of the fibroblast to the control of cancer.—In elaborating the technique for vesical carcinoma, a comparison of the methods of approaching the tumour must be carefully studied, sepsis controlled and the difficulty of determining the sensitiveness of the individual's cell realized.—Possible damage to the ureter and the iliac vessels and other complications of radium applications must also be considered.—It is possible that chemical substances which give off secondary radiations may prove of great assistance in association with radium treatment.—A record is given of twelve cases which have been treated since 1925.

Fifteen years ago the study of radium-therapy in this country was in its infancy, the medical profession, with few exceptions, having no knowledge of the results which had already been obtained on the Continent. Even America, so responsive to all that is new in therapeutics, had, likewise, lagged behind. To Finzi must be given the credit of being the first in Great Britain to use radium in any large quantity for the treatment of malignant disease. The lack of interest shown by the hospital authorities was remarkable, and it was not until the news of the disappearance of tumours when exposed to radium irradiation had filtered through to the general public that they were stirred from their lethargy. Then came the reaction, and the demand for the rare and expensive element became an urgent problem.

There followed the usual attempts of impostors to sell metal tubes which were stated to contain radium. I recall two instances, one in which a tube supposed to contain 20 mgm. of the salt proved to be empty, and the other in which the brother of a well-known gynæcologist, suffering from carcinoma of the stomach, had been persuaded to take, by the mouth, radium in solution, at the price of 10s. 6d. per bottle. How many bottles he purchased I do not know, but the one that came into my possession contained only a coloured fluid.

My acquaintance in 1913 with certain well-known workers in Paris, such as Degrais, Domenici and Becquerel, caused me to make a special study of the changes which occur in malignant tumours on exposure to radium. The findings from this investigation were incorporated in a paper read before the Section of Pathology of the Royal Society of Medicine in the spring of 1914. They entirely confirmed the work of Domenici, and may be summarized as follows :—

(I) The greatest effect upon a tumour when exposed to irradiation is produced by burying the tube containing radium in its substance.

(II) Unless the malignant cell is destroyed at the first exposure, its power of resistance is increased with each succeeding one.

(III) There are degrees of sensibility of different types of malignant cells to irradiation.

(IV) The nucleus is the first part of the malignant cell to show changes.

(V) The action of radium in the connective-tissue cells shows its similarity to the attempt of Nature to arrest the growth of cancer through overgrowth of fibrous tissue.

The most important advance since this date, in our knowledge of the changes in cells, both normal and malignant, is that made by Dr. Canti with his cinematograph. He has demonstrated that the fibroblast is not destroyed by a comparatively large dose of radium, whereas certain other normal cells are; a very valuable observation when we consider the relationship of these normal cells to the inhibition of the growth of malignant disease in the human body. The clinician is well aware of the variety of mammary carcinoma known as the atrophic scirrhus, and the pathologist

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is equally well aware why it is atrophic. The overgrowth of fibrous tissue in and around the tumour has terminated once and for all the activities of these malignant cells. Let us briefly refer to the laboratory experiments which confirm these clinical findings.

Drew, of the Imperial Cancer Research Fund, carried out a number of experiments upon the growth of cells outside the body, and demonstrated that a malignant tumour will take on disorderly growth in the absence of fibroblasts. If the latter cells be added to the culture of carcinomatous tissue, the spread of the mass is immediately restrained. In other words, the fibroblast is the constable of the cancer cell.

The data thus collected must have an important bearing on our endeavours to eradicate cancer with the aid of radium. Even though the malignant mass cannot be caused to disappear—the ideal at which all of us aim—we know now that it is possible to reduce it to impotence by bringing the fibroblast to our aid. As previously stated, a comparatively large dose of radium not only fails to have a lethal effect on this connective-tissue cell, but, under certain conditions, seems to stimulate its reproductive powers.

With these facts before me, I have elaborated a technique for the radium treatment of vesical carcinoma. To place one or more tubes in an haphazard way into a portion of a bladder growth, irrespective of its anatomical position and its degree of activity, is a common mistake which has brought discredit on the value of radium as a therapeutic agent. Its application cannot be carried out by any rule of thumb. The growth must be carefully defined, not only from within the bladder but from without. The relationship of adjacent structures, such as the ureter, rectum, and large blood-vessels should be noted, and finally an attempt may have to be made to sweep away that most serious of all cancer complications—sepsis—before treatment is commenced.

As regards the ureter, care has to be taken that it is not constricted by the overgrowth of fibrous tissue resulting from excessive irradiation in its vicinity. The same applies to the iliac vessels. Also, on account of the sensitiveness of the lining endothelium, thrombosis is a possible complication. I have seen death take place three days after the application of a tube containing 92 mgm. to within 5 mm. of the inferior vena cava. Thrombosis had extended along a considerable part of this vessel.

Access to the tumour is obtained either through the urethra (the trans-urethral method) or by performing a suprapubic cystotomy.

For the trans-urethral application of radium an operating cystoscope is required, which allows of the accurate insertion of the container into the substance of the growth. The cystoscope is then withdrawn and the radium, attached by a holder, is left in position for as long as the surgeon considers necessary. It does not require much imagination from those who have no profound knowledge of the use of radium in malignant disease to realise that treatment with the aid of the cystoscope has its limitations. There are some authorities who insist that access through the urethra is the route of choice. The treatment of any bladder disease with the help of the cystoscope requires not only a well-developed visual sense, but also plenty of common sense. Is it conceivable that even in the hands of the expert cystoscopist it is possible to determine accurately the extent of a cancer? When hæmorrhage or infection is severe, he will get merely a glimpse of the tumour. Again, the size of a tumour, as seen on the mucous surface of the bladder, is no guide for estimating the depth and width of invasion of the muscle layers.

In favour of trans-urethral application is the fact that the patient requires no anæsthetic and no incision through the bladder wall. These are its only advantages. As already stated, it is essential to define accurately the limits of the growth. This

can only be done by a suprapubic cystotomy. The technique in detail is as follows:—

Under anæsthesia, either general or regional, a catheter is passed by the assistant and the bladder washed out with an antiseptic fluid—it is my custom to use 1 in 8,000 oxycyanide of mercury—and then distended. The catheter is withdrawn and the urethra compressed by a penile clamp. With the patient in the Trendelenburg position, the bladder wall is exposed, the bladder is emptied of its fluid contents by trochar and cannula, and excised, and retractors are inserted. This allows of a thorough inspection by sight and touch of the mucous surface of the tumour. It is important to note whether the growth is of the ulcerative type or of that known as papillary carcinoma, for the method of application of the radium tubes is different in the two cases.

The retractors must now be withdrawn and the fingers of one hand passed between the outside of the bladder wall and the bony pelvis, the fingers of the other hand being in contact with the lesion on its mucous surface. Such bimanual palpation will indicate the exact position of the tumour in relation to ureter, rectum, iliac vessels and obturator nerve, also the extent of infiltation of the vesical musculature and the pelvic cellular tissue.

Having determined thus the precise anatomy of the cancer and its clinical variety, careful consideration must be given to the position of insertion of each tube of radium. Before discussing this part of the technique, it is necessary to refer to the quantity of radium and the containers which have been placed at the disposal of the staff of St. Peter's Hospital by the Empire Cancer Campaign. The total amount of radium is 200 mgm. This is put up in platinum tubes, each 0·5 mm. thick and varying in size according to the contents. There are 10 tubes, each containing 10 mgm., 1 tube containing 50 mgm., and 2 tubes, each containing 25 mgm. All the alpha- and the greater part of the beta-rays are cut off by the 0·5 mm. of platinum. For all practical purposes we use nothing but gamma radiation. The radium is kept in a safe, each tube resting in a slot drilled in a mass of lead.

One of the disadvantages in the use of radium in clinical research is the inability to alter the distribution of the element in the containers without considerable expense. There are times when one desires to use tubes, say, containing 5 mgm. each or even less. To make such an alteration may cost £60 or £70.

I now pass to the consideration of the methods and positions of insertion of these radium tubes in the two clinical varieties of bladder carcinoma. Firstly, the papillary carcinoma, the growth of the cauliflower type which has a pedicle, it may be thick or thin, consisting—apart from malignant cells—of vessels going to supply the main mass with blood. In such a case, the tubes are inserted—the amount depending on the size of the tumour—right through the pedicle, as shown in the illustration (fig. 1, *A*). The tube is left in position for twenty-four hours, thus ensuring destruction of the main blood-vessels and setting up an extensive fibrosis in the wall of the bladder adjacent to the tube, which is pushed into position by means of a special holder. The free end is threaded with silkworm-gut, and this is brought out of the bladder through the suprapubic opening and stitched to the skin. The anterior bladder wall is sewn up, except for a small opening to admit a drain and allow the silkworm gut and radium tube to be removed without difficulty, at the end of twenty-four hours. The drain is left in for another twenty-four hours.

As regards the treatment of the ulcerative type of carcinoma, the technique is modified in two respects. The centre of the tumour consists of old cells, some of which are undergoing degeneration. It is unnecessary to attack these cells, for they are senile and therefore harmless. The cells at the margin of the growth, on the other hand, are young and in active division. The radium must therefore be

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concentrated at this point and in sufficient dose not only to destroy these cells but to produce a ring of fibrosis beyond the growing edge. The tubes, then, are inserted into the wall of the bladder (fig. 1, *B*) at the edge of the ulcer, and sufficiently distant from each other to ensure that the intervening space is bombarded with a lethal dose.

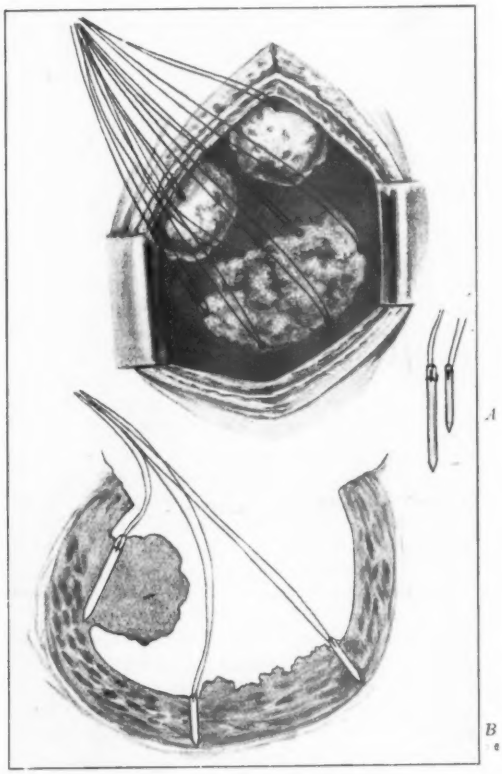

FIG. 1.—Illustrating the methods of insertion of radium tubes in (*A*) papillary carcinoma, (*B*) ulcerative variety of carcinoma.

Herein lies our greatest difficulty in radium-therapy. That which is a lethal dose for the cancer of one patient is not necessarily lethal for the cancer of another. Until we are able, by some means, to determine the degree of sensitiveness of each individual's cells, treatment must remain on empirical lines.

The further modification in technique consists in attacking the tumour from outside the bladder. Tubes must be buried in the cellulo-fatty tissue which is situated around the ureter and iliac vessels. They are threaded with silkworm-gut like those within the bladder, and are brought out on to the surface of the skin through the same suprapubic opening.

Of recent years attempts have been made to destroy cancer cells by the injection into the blood-stream of certain chemical substances, more particularly heavy metals, in colloidal solution, e.g., colloidal selenium and colloidal lead. So toxic, however, are these solutions, that with experience of their use it becomes increasingly difficult to believe that, to quote a racing expression, they will "stay the course." On the other hand, it is conceivable that if used as activators in conjunction with radium, namely, for the purpose of giving off secondary radiations, they may play an important part in a sustained attack upon a malignant tumour. Mottram, of the Radium Institute, has already published the results of experiments conducted with lead on these lines.

If a substance is used which is fixed in the malignant cell and is *non*-toxic, unlike lead, etc., our chances of success are improved. I believe it was Wassermann who first demonstrated that the coal-tar product, eosin, could become fixed in a carcinoma cell. A short time ago I was much impressed with the results of treatment of a case of carcinoma of the male breast by the injection of sodium fluorescin followed by an X-ray exposure. The investigator was Dr. Monckton Copeman. Fluorescin is a derivative of eosin, it is non-toxic, is fixed in the carcinoma cell, and will give off secondary radiations. Profiting by the experience of Dr. Copeman, I injected a malignant growth of the bladder with a 2 per cent. solution of sodium fluorescin, and at the same time inserted a number of tubes of radium. The result has been encouraging. From a scientific standpoint, however, this one case must be considered as of little importance unless the experiments are repeated over and over again.

It is a striking fact, confirmed by numerous observers, that bad results are to be expected from radium irradiation when sepsis has intervened upon a malignant growth. I know of no exception to this rule. It is worse than useless in the case of vesical carcinoma. Every effort must be made with the aid of lavage to reduce the infection to a minimum. In a foul-smelling cystitis there is no better disinfectant than a weak solution of Sanitas (1 drachm to the pint). Prognosis is far from hopeful, even when some success attends the attempts to eradicate sepsis, should radium irradiation be persisted in.

What then can be regarded as a favourable factor, apart from the size of the tumour and the type of cell? It is hæmorrhage. Patients utterly exhausted from excessive bleeding and in imminent danger of losing their lives from this complication alone, may be restored to a state of health which permits of a breathing space to allow of further efforts to control the disease. In severely bleeding growths, if the dosage and technique advocated in this paper be adopted, namely, a large quantity of radium with a short exposure, the following results will be obtained. Hæmorrhage will cease within twenty-four hours of the commencement of irradiation, in some cases within four hours. The suprapubic wound will heal rapidly. The general condition of the patient will steadily improve, and discharge from hospital may be expected within three weeks from the onset of treatment. This is what may be termed the immediate result of radium irradiation.

The remote results are as follows: In favourable cases the general condition of the patient is good, as judged by the increase in weight. Even though the urine may be quite clear, there is frequency of micturition present, due to radium reaction in the mucous membrane, and slight discomfort on defæcation resulting from irritation of the rectal mucous membrane. Cystoscopy shows, at the end of three months, a curious bullous œdema of the area of mucous membrane irradiated (fig. 2). In other cases, more particularly the ulcerating variety and when the tumour is of large size, a mass of dead growth surrounded by a raised edge, covered by what looks like normal mucous membrane, is observed (fig. 3).

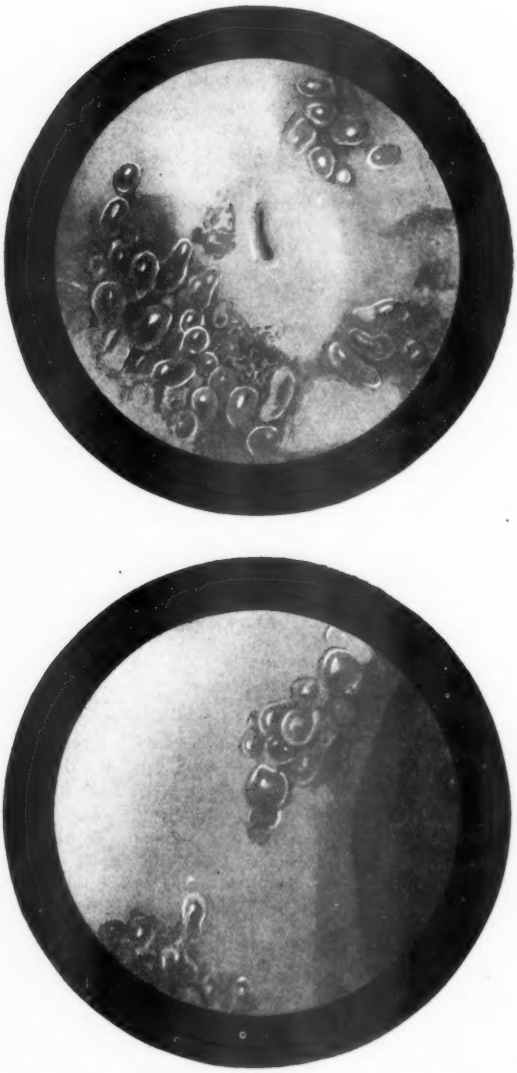

FIG. 2.—The appearance of the mucous membrane of the bladder three months after irradiation of a malignant growth.

It is common knowledge that healing of tissues following exposure to radium irradiation may be slow. With the exception of a case treated in 1925, the reverse has happened in my cases. This is entirely due to the use of comparatively large quantities of radium with an exposure of not more than twenty-four hours. The report of the Medical Research Council for 1926, on the medical uses of radium, records the result of the treatment of rectal cancer by 20 to 48 mgm. of the element, left *in situ* for periods up to ten days. The statement is made that delay in the healing of the wounds has been a noticeable feature in all cases. There is also, I submit, a grave danger of sloughing of normal tissues.

In an article in the *British Journal of Radiology*, in 1927, I drew attention to the methods of dealing with the ureter when constricted by growth, by transplanting it into a growth-free part of the bladder. With further experience, one wonders whether this is necessary provided no radium fibrosis is produced around the duct. Will not relief of pressure result from the absorption of dead carcinomatous tissue? A reference to the notes of my cases tends to support this view. Stress must be laid upon the experimental nature of the work which has been undertaken in connexion with the cases treated at St. Peter's Hospital. The radium has been applied for the most part to patients already exhausted by complications and the failures of operations or diathermy. The disease has come under treatment in the inoperable stage, and in one or two cases the cancerous cachexia has been profound.

Of twelve cases treated since 1925, four have been complete failures.

Of the remaining eight, one was first treated two and a half years ago. The patient was a man, aged 64, who had a large ulcerative growth of the right lateral wall spreading to the internal meatus. Repeated applications of diathermy seemed merely to increase the hæmorrhage. On admission to hospital he was collapsed from the excessive bleeding, and blood transfusion had to be carried out. The bladder was then opened and half the growth excised, the remainder being surrounded by tubes of 10 mgm. of radium. The patient was discharged twenty-one days later with a healed wound and passing a clear urine. For two years he enjoyed good health, but in January, 1928, there was a recurrence of hæmorrhage. Cystoscopy showed a growth involving trigone and both lateral walls. 110 mgm. were implanted into the growth for twenty-four hours. The patient was discharged twenty-one days later with wound healed but urine cloudy. March, 1928, cystoscopy showed dead growth right lateral wall and right half of base of bladder. No hæmaturia.

In three cases treated about a year ago, the patients are alive and in fair health.

One had been attending the genito-urinary department of a general hospital for three years on account of hæmorrhage, and had had numerous applications of diathermy. The growth involved the whole of the left lateral wall, including the left ureteric orifice. 200 mgm. of radium were inserted for twenty-four hours. The patient was discharged twenty-two days after operation with the wound healed and passing clear urine. This patient has put on more than one stone in weight. On March 31, cystoscopy showed a slough on the left (illustration) lateral wall, with rolled-over edge (fig. 3). The bladder capacity is normal. There has been no hæmaturia, but the urine contains a small quantity of pus.

The second case is a partial failure, in that the patient has suffered severely from radium reaction. There was much hæmaturia on admission, which diathermy had failed to control. 110 mgm. of radium were inserted into the growth, which occupied the whole of the left lateral wall and was covered by slough and phosphates. In March cystoscopy showed no visible growth, but cystitis was present and the bladder was very irritable to distension. There were also rectal tenesmus and tenderness of mucous membrane on digital examination.

The third case was that of a man, aged 63, who had been operated upon previously and treated by open diathermy for a growth of the left lateral wall. When the bladder was opened for the second time, on account of hæmorrhage, an ulcerative growth was found involving the left half of the internal urinary meatus and the greater part of the left lateral

FIG. 3.—The appearance of the bladder as seen through the cystocope three months after treatment with radium of an ulcerating growth of the whole of the left lateral wall. Note the raised edge surrounding the mass of dead growth.

FIG. 4.—Bullous œdema of the left half of the internal meatus of the bladder following radium treatment for carcinoma.

wall. 180 mgm. of radium were implanted around the margins of the growth and left in for twenty-four hours. The patient was discharged from hospital twenty-one days after operation, with the wound healed and passing urine slightly clouded, but with no blood.

On April 28, 1928, I cystoscoped this patient and found no growth, but there was bullous œdema of the left half of the internal meatus (fig. 4). The general condition of the patient was good and his weight had increased. The urine contained a small quantity of pus. He complained of irritation in the anus.

The remaining four cases have been treated within the past six months. The disease in all was in an advanced stage. In two the bladder has been opened at least twice for open diathermy. When I inserted radium tubes the prominent symptom had been continuous hæmorrhage. Within twenty-hours in each case hæmorrhage ceased. These four patients, one a woman, are alive and in fair health.

As a result of observations extending over a number of years, two definite statements can be made. Firstly, vesical carcinoma, in our present state of knowledge, cannot be cured by radium irradiation, but a shrinkage of the tumour, even to apparent disappearance, may be expected. Secondly, severe hæmorrhage can be controlled, and therefore an improvement in the general state of health will follow.

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Discussion.—Sir JOHN THOMSON-WALKER said that he would like slightly to modify Mr. Morson's statement that radium had no destructive effect on fibroblasts. That effect was, he thought, a matter of dosage, in all tissues. The difficult point about gamma-ray treatments was the small margin between the lethal effect on malignant cells and that on normal cells, and the possibility of stimulating the malignant cells if they were not completely killed.

He did not think there was any method in the case of radium of estimating what reaction the patient's cells would have to the gamma-rays. The estimation of the effect on the blood of strong X-rays, might be used, as Knox had suggested, as a measure of the resistance of any particular patient.

The striking thing about radium was, that when applied to surface growths it was very frequently successful, yet when applied to deep growths it was often unsuccessful. He remembered, however, two bladder cases in which it seemed to have had a good effect. In one (at the Radium Institute) the bladder surface was covered by papillomatous growths, and had been treated by radium introduced by means of a catheter. Concluding that no further benefit would result from applications of radium, he had admitted the patient to St. Peter's Hospital for operation. There was a delay of six weeks before her admission. When the bladder was opened, a peculiar yellow membrane of the size of the palm of the hand was found at the base, but no papillomata. The membrane was a slough on a radium burn, and it was three years before the ulcer healed.

The other case sent from the Radium Institute was of early malignant growth of small size on the posterior wall of the bladder. Into the growth he implanted a number of small radium needles, through the guidance of the cystocope, and five or six such applications were made. There was a good deal of reaction after each application, the patient became tired of the process, and demanded open operation. The growth now appeared to cover a considerable area, but on removal it had a surface of less than that of a threepenny-piece. On the other hand, he had seen cases in which, after the application of radium, the patient went from bad to worse.

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With regard to the important question of sepsis. One could not expect a case of advanced malignant growth in the bladder to be free from infection, and it was almost impossible to get rid of sepsis without removing the malignant growth. Therefore, if radium treatment was excluded when sepsis was present, the number of cases in which that treatment was available would be considerably reduced. A very important point to discuss was the position of radium "in the open market" in comparison with surgery and other treatments. In all the cases with which Mr. Morson had dealt, he (the speaker) understood that the patients had previously either been operated upon or had some other unsuccessful treatment. If radium was to be dealt with fairly, it should be tried in the same class of cases as other methods of treatment. Personally, he thought that it was not in the same category as operation for early, or moderately early, growths of the bladder, whether "simple" or malignant. In the case of a growth which could be freely excised, he would not advise radium instead of operation.

A further question was whether by the use of radium one could so reduce the size of a large malignant growth as to render it operable? He had tried to do so with a number of growths, and he was certain that one could not satisfactorily operate after radium applications. In his opinion, all kinds of irradiation reduced the patient's resistance to sepsis, and the infection increased as a result of the treatment.

Again, when excising malignant growths of the bladder, one of the chief methods of defining the incision in the bladder wall was the sense of touch. When radium had been applied, the touch became different and was no longer a guide for incision.

Mr. CYRIL R. NITCH said he understood that Mr. Morson favoured a large dose of radium, in one case 190 mgm. He (the speaker) thought the modern technique was to give a small dose over a long period. For instance, if it was desired to give 4,000 mgm.-hours, the tendency now was to give not 400 mgm. for ten hours, but 10 mgm. for 400 hours. In treating cancer of the rectum, which was almost as difficult to treat as carcinoma of the bladder, Sir Charles Gordon-Watson implanted needles of 1 and 1·5 mgm. for from five days to a fortnight. He (Mr. Nitch) had decided to treat prostate cases on those lines, i.e., by small quantities for a long period.

Mr. FOWLER WARD asked what happened to the glands in cases of carcinoma. Two years ago he had removed a carcinoma from the bladder, and transplanted the ureter. The patient had done well until the end of 1927; recently he had had threatened obstruction and enlargement of glands, with not much infiltration of the bladder. He (the speaker) would like to know what would be the effect of radium on the glands affected.

Mr. FRANK KIDD (President) said that until 1922 he had not been impressed with the results of radium applied directly, either through the urethra or through a suprapubic wound, to bladder growths. It usually served to allay hæmorrhage, but seldom appeared to cure any particular growth. In that year (1922) he had visited America and observed the work of Keyes and Barringer at the Memorial Hospital, New York. These workers were opening the bladder, destroying bladder tumours as far as possible with electric coagulation, and implanting into the charred bases glass capsules containing radium emanation.

At first he himself had worked with the bare glass tubes, which gave off not only the curative gamma-rays, but also a large quantity of beta-rays which damaged healthy tissues and were the chief cause of slough formation. Later on he had employed the method of enclosing the bare glass tubes in platinum screens of a thickness of 0·3 mm. These shields filtered off 98 per cent. of the beta-rays, but did not inhibit in any way the action of the gamma-rays on the neoplastic cells. If a platinum-screened glass seed containing 2·5 millicuries of radium emanation was implanted into the tissue of a growth, it appeared to be capable of killing off all the tumour cells within a radius of 1·5 centimetres. Such a seed lost about half its effective rays in ten days' time, and in the course of some thirty days it had given off all its effective dose. These seeds could be buried in the tissues and left there indefinitely (as proved by the radiograms exhibited) without appearing to do any harm at all. Nevertheless, he was now accustomed to attach threads to the capsules by means of which they could be removed at any time, if so desired, either cystoscopically or through a suprapubic sinus. Since employing the platinum screens he had had practically no trouble with sloughing and sepsis.

He had, nevertheless, gained the impression that it was possible to do too much with radium. It was better to do too little and repeat the dose, rather than do too much and get extensive sloughing. This was particularly the case in dealing with bladder growths, when the sloughs must of necessity be bathed in urine, and were likely to become infected and lead to slow healing of bladder wounds with the risk of ascending pyelonephritis. It was better not to introduce more than eight, or at most ten, platinum seeds, each containing 2·5 mc., at one sitting. He had had success with more, but had also had failures from sloughing and sepsis. Doubtless there was a wide field for employment of radium in the treatment of bladder growths, but the exact indications and the technique were still in the process of modification and improvement.

In some cases he had destroyed a tumour of the bladder by means of cysto-diathermy, and had implanted radon seeds into the charred base by means of a cystoscopic implanter. In the majority of cases he had exposed the growth by means of suprapubic cystotomy, taking the utmost pains to avoid any step in technique which would be likely to lead to implantation of seedlings of growth on the suprapubic scar. The technique was so exact that so far he had never encountered this unfortunate complication, a fertile source of trouble in many reported cases.

He could report twenty-three cases, since the end of 1922, in which he had attacked tumours of the bladder with electric coagulation followed by the implantation of radium emanation seeds.

There had been twelve cases of malignant papilloma single or multiple. In six he had carried out treatment entirely through the cystoscope, using his own new diathermy cystoscope (described in the *Lancet*, December 19, 1925, p. 1282), followed by radium implantation through a special cystoscopic implanter. Five patients yielded good results and were apparently well. One lived some years without symptoms but finally died of recurrence at the age of 87. In six he had opened the bladder and carried out the manipulations under free observation. All of these cases had done well to date. The open method seemed to be the method of choice in doubtful and difficult cases; the results were more certain and the procedure possessed little additional danger.

There had been eight cases of papilliferous carcinoma. Three of these were treated by the closed cystoscopic method; all were in women. In two the results were remarkably good, one having kept free from recurrence since 1922. In the third case the bladder had to be opened and treated a second time, with favourable results so far. Six cases had been treated by the open method, and three were at the present time free from recurrence. In two there had been recurrence and the patients had died within from six months to a year after operation. One had died within three months from uræmia and anæmia, probably as a direct result of the sloughing caused by the operation.

There had been three cases of ulcerating carcinoma treated by the open method. The patients had all died within a short time and the treatment had done nothing except delay the end.

Partial cystectomy held out the best hope of permanent cure in cases of malignant disease of the bladder. He had a number of patients alive and well from twelve to sixteen years after that operation. Unfortunately, partial cystectomy could not be always carried out owing to the position or size of the growth. When the growth was situated below the ureters, between the ureters and the internal meatus, or below the ureters and somewhat laterally to the trigone, partial cystectomy should not be attempted, but the cases should be treated by opening the bladder, destroying the growth with diathermy and implanting a sufficient number of radium emanation seeds according to the size of the growth. There was another type of case in which this method seemed valuable in helping to prevent recurrence, that of single or multiple malignant papillomata too large for cystoscopic treatment, in which the bladder had been opened for inspection of the growth which was found not so far advanced as to warrant a partial cystectomy. In some of these cases, at the operation he decided not to employ cystectomy but to burn away the growth and implant radium.

Radium, though it could not replace surgery, would take its place as a valuable adjunct in attacking bladder growths. Its use could only be learnt by experience, and no doubt many improvements in technique would occur during the next decade. He appealed to urologists in this country to pay close attention to radium.

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Mr. CLIFFORD MORSON (in reply) said that those who had seen Dr. Canti's cinematograph demonstration showing how periosteal fibroblasts had remained apparently unaffected when attacked by a very large dose of radium, would be convinced as he (Mr. Morson) was that these fibroblasts were the most resistant of all cells to the action of radium, and that a much larger dose than had yet been used would be needed to destroy them.

Sir John Thomson-Walker had well said that the whole problem was what constituted a lethal dose for the malignant cell, and what for the normal cell. The lethal dose was probably different for different people, and no radium worker had yet evolved a scheme for giving the necessary data. He was diffident about using radium in a case in which he thought he could remove the whole growth by partial cystectomy; it was a serious matter to experiment with treatment other than operative for a small growth of the bladder. His experience with regard to radium and malignant disease of the bladder was increasing, and before long he would venture to treat an early case.

He agreed that it was of no use to treat these cases with radium and then attempt partial cystectomy; the radium devitalized the tissue, and therefore the results of operation were bad.

With regard to Mr. Nitch's remarks concerning a small dose extended over a long period being preferable to a large dose for a shorter time: in a discussion recently held by the Section of Electro-Therapeutics, several speakers said they were not now using small doses over long periods, but were giving a comparatively short exposure with a larger quantity of radium. And in the Report of the Medical Research Council for 1926 it was stated definitely that small doses of radium over a long period devitalized the tissues more than did larger quantities over a shorter period. There had been very unfortunate results in rectal cancer when small quantities of radium had been left *in situ* for ten days. The tissues had refused to heal, even though ultra-violet rays had been used to try to increase their vitality.

In answer to Dr. Fowler Ward's remarks as to metastasis, he (the speaker) was sure that radium was purely local in its action. In the case of the patient with a growth on the tonsil and enlarged glands in the neck, the swelling in the glands had subsided when the growth disappeared. He had once thought that the result was due to absorption of some chemical substance reacting on the cervical glands, but now he felt sure that the radium put into the throat acted directly on those lymphatic glands and shrivelled them up; he felt equally certain that radium had no effect on distant metastases.

He had been much interested in the President's remarks and in the results which Mr. Kidd had obtained with radium. But one point against the seeds charged with radium emanation was that they were really foreign bodies in the bladder. If they were going to destroy the malignant growth they would, in due course, fall out into the bladder, and in time would be coated with phosphates, and so cause further complications. [The PRESIDENT: I have put in over 200, and that has not happened.] He knew that American surgeons were enthusiastic as to the use of these seeds, but he (the speaker) had never had the same success with seeds as with the radium salt. He believed the main reason was that the strength of the emanations gradually decreased, while with a tube containing the actual radium salt the maximum effect was being obtained during the whole time, therefore a lethal dose was operative so long as the tube was *in situ*.

Sections of Tropical Diseases, Psychiatry, Neurology and Balneology.

[*March* 13, 1928.]

DISCUSSION ON NEUROSES IN THE TROPICS.

Dr. H. B. Day.

THIS subject may, for discussion, be usefully considered under two headings: (*a*) The neuroses or psychoneuroses as seen in the native inhabitants of tropical countries; (*b*) the effect of tropical conditions on the development of neuroses in European residents.

The study of the mind of the native in the primitive races of mankind, and in the different and aged civilisations of the East, is of fundamental importance in elucidating the mechanism of the psychoneuroses. Even the most primitive peoples have an elaborated social code, involving restrictions and subordinating the interest of the individual to the general welfare. With the development of a higher standard of knowledge and civilization we find an increased demand on the powers of inhibition and control. Let me take one instance of political importance—that of free speech. The success of parliamentary or democratic government depends on the free expression and interchange of opinion, proceedings which impose a measure of forbearance on the part of those whose own opinions may happen to differ. To introduce such institutions to a race long accustomed to autocratic rule, is to invite pandemonium.

In unsophisticated natives hysteria is relatively common. Indeed, the mass effect of emotion and suggestion is regularly exploited by tribal songs and war dances. In religious ecstasies the devotees are wrought to a condition of hypnosis and perform acts which could hardly be attempted in cold blood.

That hysterical manifestations are amenable to suggestion is recognized by the Egyptian peasantry. Whereas medical treatment is commonly sought in cases of ordinary illness, for the hysterical patient a special religious ceremony is generally convened.

Psychasthenia occurs among races, as well as persons, of a neurotic type. In the East it often appears to be an undesirable waste-product of competitive conditions introduced by western civilization. The mental strain is too great for the weakling, and a nervous breakdown may occur if the additional load of a toxæmia or conflicting emotions and thwarted hopes be imposed.

Similar and further strains affect the European resident in the tropics. The monotony of trying climatic conditions and of isolation are in striking contrast with life here in England. When scorching days are succeeded by suffocating nights of heat, loss of sleep often prevents the restoration of nervous energy. The tension of an electrically charged atmosphere or the monotonous downpour of tropical rain —as dramatized in a recent play—are equally destructive of rest. The petty annoyances of life in the tropics—the irritation of biting insects, uninviting food and the necessity of holding communications in a foreign language, often imperfectly understood,—have a cumulative effect. Under these circumstances a condition of true neurasthenia—nervous exhaustion—may develop. Everyone has a breaking-point, and in the endeavour to avoid failure, the victim may have recourse to immoderate use of alcohol with direful results. The effect of such climatic strain is too well known to need elaboration. Petulance over trifles, a hair-trigger temper, increased reflexes, a blunting of the finer qualities and mental inertia reveal the condition.

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To counteract the harmful effects of tropical conditions, suitable hygienic measures and regular periods of leave are recognized precautions. In the case of men taking appointments in the tropics, we have the additional safeguard of careful selection and apprenticeship, the interest of new work to be done and the frequent necessity for physical exertion. These are often lacking for the women, for whom outside distractions are few and the scope of domestic work is much restricted. As has been well said, it is on the women and children that the "white man's burden" falls most heavily.

The development of a psychoneurosis in an individual is due to his failure to adapt himself by mental adjustment to the circumstances of his environment. With the complete change of surroundings that residence in the tropics entails, a considerable call is made on the adaptive powers of the European. The majority effect a settlement, and the mental activities and desires find satisfaction by new outlets.

Some, however, fail to do so. Persons whose moral control and education are not of high degree may succumb to the temptations of their new environment and gradually sink to a low level. They satisfy their desires at the price of their civilization. The same fate may befall those of good promise, whose higher mental faculties have been blunted by alcoholism or by repeated illness.

In others an unresolved mental conflict persists and may lead to a psychoneurosis. Although the symptoms of these disorders are so varied, I know of no single one that is peculiar to residents in the tropics.

So far I have touched on some of the outstanding racial, climatic and individualistic elements that are concerned in the production of neuroses. There remains an important and interesting factor—the part played by tropical diseases. Certain of these are capable of inducing not only psychoneuroses, but actual insanity. I refer particularly to malaria and pellagra.

As regards the responsibility of malaria for racial degeneration and for mental disorder in individuals, a most interesting work has been written by Anderson.[1] In his experience it has been the subacute, chronic forms of the disease dragging on for months without effective treatment that have commonly been responsible for mental derangements. As with other infections, a confusional insanity or melancholia is the usual form of psychosis produced by gross cerebral involvement. Psychasthenic conditions are more common, characterized by mental apathy, inattention and amnesia. The patients may have no recent history of chills and fever, or spleens sufficiently enlarged to be palpable. For diagnosis Anderson recommends a provocative injection of adrenalin to mobilize parasites.

Pellagra, though considerably restricted in its geographical distribution as compared with malaria, is a more important cause of nervous disease. Of adult pellagrins, about 40 per cent. show mental deterioration. The effect on the brain appears to be a purely toxic action on the neurones, and the symptoms range from simple depression and backwardness to confusional insanity and melancholia, terminating in dementia. The exact causation of pellagra is still in dispute, but there is no doubt that gastro-intestinal disorder is a very early symptom, and that troublesome diarrhœa is frequent in advanced cases and may prove fatal. In such cases I have found the stools highly acid, and often fermentative, as in sprue.

Indeed, intestinal infections appear to be an important cause of psychoneuroses and states of depression. The sufferer from chronic dysentery or sprue can rarely preserve a cheerful disposition. We find the same tendency to depression, however, when the intestinal symptoms are not severe, as in chronic amœbiasis. In this country the association of colitis with neurasthenia is notorious, and a streptococcal

[1] Anderson, W. K., "Malarial Psychoses and Neuroses," London, 1927.

infection of the intestine appears responsible for the development of not a few cases of psychasthenia, and even of melancholia.

In conclusion, therefore, I would urge that the victim of neurosis in the tropics be regarded as a sick man, and that every effort be made to discover any infection to which he may have been exposed in the tropics, since this may manifest itself in the development of a neurosis.

Lieutenant-Colonel BERKELEY HILL, I.M.S.

In 1926, a correspondence in the *British Medical Journal* arose out of a letter from the Bishop of Singapore, entitled "Mental Irritability and Breakdown in the Tropics," inquiring as to the cause of the upset of mental balance which is so common in the tropics and ranges from excessive irritability or loss of judgment to lunacy or suicide.

To this inquiry the Journal published eight replies, mostly from medical men. Now, while all the writers admitted that "upset of mental balance" was common amongst Europeans in the tropics, no two agreed as to the cause. Among the causes cited by these doctors were: altitude, moisture, strong sunlight, eye defects, worry, hyperæmia of the brain, north wind, barometric pressure, electrical content of the atmosphere, food, drink, constipation, native servants, masturbation, and smoking. Hardly anything could show more clearly than these replies how almost hopelessly ignorant we are about a state of affairs which no one can deny to be of considerable importance.

That the laity has long recognized that Europeans are more liable to mental disorder in the tropics than in temperate climates, is shown by the existence of such terms as "Punjab head," "West Coast head," "Soudanite," "Colonite" and "Tropenkoller." Further, that this tendency to mental upset is no product of modern life is shown by the letters of Sir Arthur Wellesley (afterwards the famous Duke of Wellington) to his brother, Lord Mornington, in which he deplores the frightful irritability of many of his British officers in the Army in India. Novels dealing with life in the tropics abound in references to mental disorder among Europeans. Kipling's "A Disturber of the Traffic," Conrad's "An Outpost of Progress," and Seton Merriman's "With Edged Tools," all contain vivid descriptions of such disorders.

Reference to these conditions in medical literature is scanty. In the "Handbuch der Tropenkrankheiten" edited by Dr. Carl Mense, Van Brero, a Dutch neurologist, discusses what he regards as a specific form of irritability among Europeans resident in the tropics, under the name of "Tropenkoller." Van Brero admits that "Tropenkoller" is rare among Europeans who live under agreeable conditions but common among those who live lonely lives deprived of the usual amenities of civilization. He considers the climate a contributory factor and when loneliness and discomfort are added, a neurosis develops which leads to excesses in Baccho et Venere with their inevitable consequences of mental degeneration. Van Brero concludes his short but interesting review of this important phenomenon by reminding his readers that in our present ignorance of tropical psychopathology we must suspend judgment as to the cause of these psychic disorders. Besides this so-called "Tropenkoller," Van Brero specially mentions insomnia among Europeans in the tropics, particularly at the change of the seasons, and he ascribes to want of sleep a considerable share in the production of neurotic disorders. Scheube, in his "Krankheiten der Warmen Lander," believes that long residence in a tropical climate produces sleeplessness and "nervous irritability," and the latter, when coupled with chronic malaria, ends in neurasthenia. Scheube considers mental disorder more common among Europeans in the tropics than in their own countries. He holds

strongly to the view that no one of a "neurotic" stock should embark upon a career involving prolonged residence in the tropics. Like Van Brero, Scheube cites "Tropenkoller" as one of the prominent psychic disorders of Europeans in hot countries, and he directs attention to Mense's opinion that this morbid emotional state is a remarkable compound of hate and affection; a view which agrees with the Freudian conception of the ambivalence of these emotions. Mense goes so far as to hold that a considerable percentage of Europeans who elect to dwell in the tropics are by nature "eccentric," and it is owing to an inherent abnormality of temperament that they leave their own homes! Although this view may not make a strong appeal to many people, especially the English, my experience has led me to believe that there is a good deal of truth in it.

We may turn now to a lecture by Colonel H. W. Acton, I.M.S., published in the *Indian Medical Gazette*, 1927, on neurasthenia in the tropics. Among the numerous symptoms which, in his opinion, make up the clinical picture of this form of neurasthenia, irritability is not included. On the contrary, Colonel Acton considers that the chief characteristic of tropical neurasthenia is depression, with a tendency to hypochondria or morbid anxiety. In discussing the ætiology of the condition, he classifies the causes as exciting and predisposing, toxæmia being pre-eminent among the former, while he places among the latter climate, anæmia, bad food, and excessive exercise.

Now, it may occur to neurologists that if we disentangle this mass of symptomatology, we shall find that Europeans living in the tropics are prone to a neurotic syndrome, the central symptom of which is a state of hyperexcitation manifested in a general irritability or a condition of morbid anxiety.

Although hyperexcitation may appear as a symptom of any form of psychoneurosis or psychosis, none show it so consistently or so strongly as that neurosis to which Freud has applied the name anxiety-neurosis (Angstneurose).

According to Freud, the clinical picture of anxiety-neurosis comprises the following symptoms:—

(1) *General Irritability.*—Although this is a symptom common to many nervous conditions, it is invariable in anxiety-neurosis. One expression of this irritability is over-sensitiveness to noise, and auditory hyperæsthesia as a frequent cause of insomnia, more than one form of which belongs to the anxiety-neurosis.

In the medical evidence which I have cited, there is a general consensus of opinion that "irritability" and "insomnia" are prominent symptoms of mental disorder among European residents in the tropics. The same applies to auditory hyperæsthesia. Any resident in the tropics knows that whenever he is a "little run-down," the chattering of native servants, a dog barking, or a tom-tom beating, can give rise to exquisite suffering.

(2) *Anxious Expectations.*—Under this heading we may class a particular form of anxious expectation very commonly observable among Europeans of both sexes in tropical countries. I refer to anxiety about one's own health. This "hypochrondria," to use an old term, is generally manifested in relation to sunstroke, glare, and bowel complaints. Besides hypochondria, another form of anxiety appears in pangs of conscience, over-scrupulousness and pedantry. In my experience this type of anxiety is not uncommon among Government officials, especially Europeans of the Indian Civil Service, with whom it may sometimes amount to a veritable "folie du doute."

(3) *Awakening in Fright.*—As undue sensitiveness to noise may cause insomnia, so this condition of anxious expectation may evoke "pavor nocturnus." As anxious expectation is the nuclear symptom of this condition, we might say that there is here a quantum of anxiety in a free-floating condition, ever ready to attach itself to any suitable ideational content.

(4) *Brooding.*—This mental occupation is an expression of the anxiety-state, and appears fairly consistently in the syndrome. It arises from the individual's effort to disprove that he is ill, as his hypochondriacal phobia maintains.

(5) *Disturbances of the Alimentary Canal.*—In the anxiety-state the digestive processes are not the only ones subjective to disturbances, but they are perhaps the most characteristic. Nausea and biliousness are not at all rare, and probably a good deal of "liverishness" is neurotic rather than hepatic in origin. The symptom of ravenous hunger can, by itself, or in combination with others, constitute a rudimentary anxiety attack. In other cases there may be a tendency to diarrhœa, which may cause the queerest diagnostic mistakes.

From the above sketch of the principal manifestations of the anxiety-neurosis, I think there is justification for the assumption that the mental disorder of Europeans in the tropics is a species of anxiety-neurosis. The validity of this supposition is strengthened when we consider the ætiology of the anxiety-neurosis. From the investigations of Freud and others it appears that the prime ætiological factor in the anxiety-neurosis is sexual excitation without adequate paths for gratification. Such privation causes an accumulation of somatic excitation, which then tends to dissipate along paths through which it can discharge more easily than along the path to the mind. Libido will therefore subside, and the excitation will express itself as anxiety. In women the principal cause of anxiety-neurosis is "coitus-reservatus," but voluntary or enforced abstinence will have the same effect on women as on men.

I maintain that the conditions under which Europeans generally, though by no means always, live in tropical countries, promote the development of an anxiety-neurosis, especially in men.

First, sexual abstinence is frequently, for many Englishmen in the tropics, inevitable even when they are married. The seasonal exodus of European women to the hills, or to Europe, involves constant changes in their sexual lives and in that of their husbands. To both, but more especially to the man left to toil in the heat of the plains, or of the large cities, deprivation of the means of gratifying the sexual impulse cannot fail to produce some effects, direct or indirect. To the direct effects I have alluded already. From among the indirect effects I select only one, alcoholism, because it is not only the most important but by far the most common. By "alcoholism" I do not mean immoderate consumption of wines and spirits, although this may sometimes supervene, but the resort to drinking to escape from the psychic tension set up by the anxiety-state. We know that alcohol promotes the regression of the sexual libido to a more primitive level by paralysing the inhibitory forces which ordinarily hold it in check. In this instance the regression is from the heterosexual to the homosexual level. One has only to sit in the bar of any club in the tropics to witness this phenomenon. It is only necessary to watch the behaviour of the majority of the men there and to listen to their conversation to recognize unmistakable signs of the regression of libido. Still worse, of course, is the condition of the European doomed to live alone in some out-of-the-way place. Here the absence of another of his own race and class may quickly bring about a loss of self-respect, so that any degree of alcoholism, up to dipsomania, may develop. Now, because alcoholism lowers the forces of inhibition in one direction, i.e., the sexual, it does not exclude other directions also. Indeed, alcohol reduces the inhibitory forces in every direction, so that at least one other consequence is a loss of control over the emotions. Hence, it is hardly surprising that occasionally in the tropics we meet Europeans who suffer either from violent outbursts of anger or from brooding melancholy. Of course, the quantity and quality of the reaction vary with the temperamental and cultural attributes of the individual concerned.

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We must now consider the second important cause of anxiety-states—coitus interruptus. This practice is lamentably common among Europeans living in tropical countries. The bad effects of it are more often met with in women than in men, but neither sex is exempt. The effects of prolonged coitus interruptus are many and varied—sleeplessness and general irritability, with hyperæsthesia to auditory sensations, are perhaps the commonest. Other chronic symptoms are giddiness, paræsthesia—simulating rheumatic pains—vasomotor congestions and gastro-intestinal disturbances, especially nausea and diarrhœa. When one group of symptoms is especially prominent, particularly the cardiac, respiratory and gastro-intestinal, the condition is very apt to be mistaken for organic disease.

I will conclude by a brief reference to the matter of statistics on this subject. To the best of my knowledge none exist. For example, the influence of temperature, humidity, clothing, diet, work—physical and mental—exposure to the sun, isolation and many other factors, on the mental well-being of Europeans in tropical countries, remain unexplored. Even in respect to temperature—perhaps the most formidable of all the tropical conditions—very little is known. In his interesting study on the character of races, Huntington states that for all European people the most favourable conditions of climate are essentially the same, namely, a temperature which on the average for day and night, does not rise above 70° F. in summer, or fall below 40° F. in winter. Extremes may of course be greater. Physical health and activity are at their highest when the outdoor temperature for day and night averages about 64° F., while mental activity seems to be greatest at a lower temperature, possibly as low as 40° F. among people who heat their houses. A fairly high humidity appears also to be favourable at moderately low temperatures, but not at high temperatures. Variability and storminess seem also to be important. While all races have not yet been investigated in this respect, many facts suggest that for practically the whole human race an average temperature between 60° F. and 70° F. is better than one which averages about 70° F. It is therefore evident that Europeans in tropical countries, except those who live at high altitudes, exist under a perpetual disability. This being so, the climate of all tropical countries must play an important, but hitherto unassigned, rôle in the mental well-being of European residents.

Dr. Hugh S. Stannus.

Anyone with tropical experience, and to some extent anyone with experience of patients returning from the tropics, will have realized how commonly men and women, whose work lies in our tropical possessions, suffer disturbance of normal mental health. The subject has received but scanty notice because, I imagine, it was considered that no factor essentially belonging to the tropics entered into the problem; others, of course—witness the correspondence in the *British Medical Journal* quoted this evening—have laid the blame on almost every conceivable component of a tropical environment. The problem is not any the easier for lack of definition of the conditions met with.

Colonel Berkeley-Hill has attempted to disentangle "this mass of symptomatology," and comes to the conclusion that the condition in question is "a species of anxiety-neurosis," and follows Freud in believing that "the prime ætiological factor is voluntary abstinence from sexual intercourse," a factor therefore which may depend on conditions of residence in the tropics, but not due to any condition essentially of the tropics.

I became interested in the subject many years ago, and in 1926 read a paper on Tropical Neurasthenia before the Royal Society of Tropical Medicine and Hygiene: what I have to say to-night has therefore been said before.

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I look to a much wider ætiology than does the opener of this discussion. I believe that the condition commonly seen in the tropics is a neurasthenia, a neurosis consisting in "a faulty response" by overaction "to the difficulties which life offers" (Ross), the symptoms being summed up in abnormal fatiguability and irritability. The neurasthenic "begins the day tired, any mental or physical exertion being accompanied by a sense of exhaustion, with headache, a feeling of pressure at the vertex, tired eyes; walking causes aching legs, sitting gives rise to cramps; food gives him dyspepsia, and excitement prostrates him," to quote Yellowlees. He may complain of pains in the back, giddiness, hyperacusis and tinnitus, intolerance of light, anorexia, flatulence, with constipation or diarrhœa, vasomotor disturbances, cardiac discomfort and palpitation, lack of concentration, impaired memory, insomnia, etc., all symptoms of abnormal fatiguability and hyper-irritability.

Why do these symptoms occur, and what is the underlying mechanism; why do they resemble those of asthenia due to injury and disease, and what part do conditions in the tropics play? The faulty response—overaction—is, I believe, due essentially to the introduction of *fear* into the sufferer's mind, fear, whether it be of physical hurt or a component part of grief, worry, anxiety, apprehension, disappointment, etc. On the psychological side it has origin in misunderstanding and misinterpretation.

In seeking for an explanation of the mechanism of the production of symptoms, the work of George Crile is very suggestive. He has pointed out that the phenomena of fear and rage resemble those of great muscular effort, that phylogenetically these are closely related, acute fear being the emotional complement to the most extreme form of muscular activity entailed by violent struggle with, or headlong flight from, an enemy; further, that "now, whenever the emotion of fear is strongly felt, though it may not lead to any exertion, the same result tends to reappear through the force of inheritance and association; that this tendency should persist to-day, in spite of the disappearance of most of the stimuli to active physical combat, so that at the slightest hint of danger man's energies are drained exactly as in the old days of physical struggle, is one of the misfortunes of our insufficiently adapted state," the signs of exhaustion from emotional disturbance resembling those of exhaustion from muscular exertion. Crile has further produced evidence to show that a system of organs has been evolved for the specific purpose of transforming potential into kinetic energy—the kinetic system for defence—consisting of the brain, adrenal, thyroid and liver acting in conjunction with the muscles. Acute over-stimulation may lead to acute exhaustion, whether the stimulus be great physical effort, physical injury (the basis of Crile's theory of surgical shock), or acute emotional disturbance such as fear. Further, just as *acute* exhaustion may result from an acute noxious stimulus, so a summation of minor noxious stimuli, whether physical or emotional, may produce *chronic* exhaustion with symptoms of neurasthenia. Persisting over a long time, a vicious circle may be established, the stimulated thyroid lowers the threshold for incoming noxious stimuli, noxious brain-action patterns become established, the adrenals are excited to over-production, in turn activating the brain, while the liver is called upon in relation to the metabolism of carbohydrates and the detoxication of waste products. Any external factor may again act upon some link in this chain and help to precipitate the unfortunate result; thus we see the part played by infective and toxic processes, including alcoholism, as well as helminthic, protozoal and bacterial infections.

In the neurasthenia of the tropics we may see noxious physical and noxious emotional stimuli severally at work. We may see cases of neurasthenia which do not differ in their causation from cases in this country, or we may see cases due to factors intrinsic to life in some parts of the tropics.

Think of them together, all associated with fears and anxiety. The man exiled

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from home—separated from his family—in financial straits—lonely, or lacking congenial society—disappointed over promotion—unable to escape from monotony. Living amidst a native population causes him annoyance at every turn, because he has never troubled to understand its language and its psychology. From morn to eve he is in a state of unrest. Beset all day by sodden heat, and the unceasing attention of insects, he is driven to bed only to be kept awake by the reiterated cry of the brain-fever bird or the chorus of frogs. Never at rest! Always an "on-guardedness"! Always suffering from a summation of multiple, minor, defence-inducing noxious stimuli. Herein lies the cause of essential tropical neurasthenia.

A few words on climatic conditions. There is very little evidence that the sun's rays are harmful, except in so far as they may act as noxious stimuli in acute sunburn, or when they impinge directly on the retina. Excessive heat, especially when associated with high atmospheric moisture, stagnant air, improperly constructed houses, unsuitable clothing, etc., together with atmospheric and terrestrial electrical disturbances, are certainly adverse factors, as anyone who has lived in the tropics will know. They act upon the same system to which reference has been made above.

Mr. A. F. MacCallan

said that he was one of the first people who had replied to the Bishop of Singapore's letter, and in his reply he had pointed out the extraordinary strain which was placed by residence in the tropics on those who had slight errors of refraction. Often, in this country, one heard ophthalmologists speak of small refractive errors as not being of any great importance, but in the tropics an error of astigmatism of only $\frac{1}{8}$ of a diopter might make all the difference between happiness and misery to the person concerned.

But, apart from eye conditions, those who had resided in tropical and subtropical countries must have suffered at some time during their stay from neurasthenia, in varying degrees, and hence we should endeavour to ascertain whether in hot countries there was any peculiar physical basis for this trouble.

Fatigue-indigestion was extraordinarily common in the tropics, and was of great importance in the production of neurasthenia. A man went for a ride before breakfast, spent a long morning at his work, then ate an enormous lunch, and went to sleep afterwards. On awaking he felt very ill and during the remainder of the day suffered from indigestion. This was due to fatigue, which had greatly lowered his vitality.

The question of the consumption of alcohol in the tropics was one which had always received a good deal of attention. Judging from his own tropical experience, however, its importance had been a good deal exaggerated. He regarded alcohol as a very useful anodyne when used in moderate quantity; it was resorted to by many men to bring about a needed dulling of the finer sensibilities.

Dr. J. F. C. Haslam

said that Dr. Stannus was probably right in stating that tropical neurosis, or tropical neurasthenia, was really due to the total effect of a large number of noxious stimuli. He (the speaker) would go further and place more importance on purely climatic conditions than Dr. Stannus did. The idea was held by some, especially in Australia, that in purely climatic conditions there were no necessarily noxious influences at all, and that in the various tropical diseases alone were to be sought the noxious influences which made residence in the tropics difficult and harmful for Europeans. In Australia, however, had arisen the expression "kitchen neurasthenia," indicating that the form of neurasthenia commonly recognized in the tropics had been frequent among housewives who spent most of their time in the kitchen—

the place where the temperature was highest—though it had been denied that temperature and humidity were, in themselves, deleterious.

There had been many observations with regard to industries involving exposure to high temperatures and humidity, suggesting that those cotton workers, for instance, who were exposed to high temperatures and a humid atmosphere, did not show a greater incidence of disease than did those whose work lay in cooler and dry conditions in the industry. Other recent inquiries, however, had shown that as soon as the wet-bulb thermometer rose above 75° F., the efficiency of the worker began to fall off. That, and the physiological inquiries which had been summarized by the United States Public Health authorities, showed that high temperature and humidity were factors which, in any excessive degree, exerted a harmful effect on Europeans.

Dr. HALLIDAY SUTHERLAND

said that the point of interest was whether the toxins of tropical diseases could produce mental disturbance analogous to that produced by other toxins, such as alcohol. A month previously he had had a patient brought to him in a paranoid condition, who had given his name as "God the Father," and stated that he had destroyed the Universe (the same night he destroyed the electric-light fittings in his bedroom!). For eighteen months there had been ideas of reference and delusions of persecution. The mental breakdown had begun in the East, where the patient had lived for twelve years. He had suffered from malaria, amœbic dysentery, typhoid, para-typhoid B, dengue, and sand-fly fever. The cerebro-spinal fluid and the blood gave a negative Wassermann reaction. There was no agglutination to typhoid or to the para-typhoids. No malarial parasites were found. In doubtful cases it was wise to think of potassium iodide, but in the tropics they also thought of quinine. He had given quinine and thyroid in this case, also Guelpa's treatment and the Plombières douche. The patient had so far recovered that he was now analysing his own ideas of reference, and believed that twelve years' contact with natives, who used language as often as not to conceal truth, had led him to seek for hidden meanings which, as he realized later, had no real existence. Two members of the Section of Psychiatry had seen the patient in consultation. The first had agreed with his (the speaker's) interpretation and had given a hopeful prognosis. The second, who had given an unfavourable prognosis and expected progressive mental deterioration, said that, if the exciting cause had been recent alcoholism, a relatively favourable prognosis could have been given. In the East the patient had taken half a bottle of whisky a day, but for the past eighteen months had had little or no alcohol. That was why he (Dr. Sutherland) had raised the point as to whether the toxins of chronic malaria might not produce in a neuropathic subject a psychosis similar to that of alcohol. In that event a complete recovery might not unreasonably be expected.

Dr. MILLAIS CULPIN

said that an important experiment was being carried on in Australia. Before he took up the study of medicine he had spent five years in a tropical part of Queensland where the annual rainfall was 40 inches. The people there lived a very hard life. He had been in charge of a local school with an average attendance of thirty-two children. There was no doctor within seventy miles and, except for an occasional broken bone, they did not need one. Dengue fever was the only epidemic. In spite of the hard living, he did not remember to have seen anything like neurosis among those people. Later he had experience further south, and practised in Brisbane, a subtropical city, where there seemed to be no more neurosis than in this country. In tropical northern towns the people lived a family life with all their interests there. There were about 12,000 people in one town, and they carried on, as far as

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he could observe, without the kind of disorders under discussion and said to be characteristic of the tropics. He could understand "kitchen neurasthenia" because the women were tied to their kitchens. The men had more freedom; if a man did not like his job he could clear out, and usually did. He sometimes did so if he did not like the climate. It was a very different proposition with the women, and in their case trouble was likely to develop.

He had practised for five years in Shanghai. In Australia they had no tropical diseases to speak of; malaria did not count. Dysentery and sprue were rarities, though ankylostomiasis was met with. But in Shanghai there were tropical diseases, and Shanghai, though a luxurious city, was a "nervy" place. Two of his colleagues had recently expressed their pleasure at getting away from it because of the "nervy" state it produced in them. He (the speaker) thought, however, the chief factor was that people did not go there for the purpose of making their home and settling down, but life was all worry and hustle and there was no stable home life to counteract this. The manner of life was not confined to Europeans. Chinese suffered from psychoneuroses, though they did not suffer as much as did Europeans from tropical diseases. Looking at the case of Australia, one could not blame the climate. Many Australians considered that tropical Australia was a very good country to live in. Indeed, one medical officer had said that if tropical diseases could be abolished, the tropics would be the only places on earth worth living in.

Sir ROBERT ARMSTRONG-JONES

said he was more familiar with the more pronounced mental disorders of the tropics than with their earlier phases, as he had seen these fully-developed forms in the French colony of Tunis, where some years ago a modern mental hospital for 100 cases had been opened in connexion with the general hospital, and he had the privilege of being there as a delegate of the Medico-Psychological Association at its opening. The late Dr. Régis, of the University of Bordeaux, had then presented a brochure of about 200 pages on the care of the insane in the French colonies, and he included their care in the neighbouring colonies of Holland and of Great Britain. So valuable was this contribution that the French Minister for the Colonies helped to make it known to French military and naval surgeons, as well as to the colonial and civil medical staff. Cases of insanity among natives and Europeans, even in French equatorial Africa, were sent to St. Peter's Mental Hospital, Marseilles, and to Tunis for treatment, and among these, malaria figured extensively, often associated with alcoholic symptoms, cases of acute toxic polyneuritis resembling Landry's paralysis being often met. Among those races with undeveloped civilization, the most frequent forms of mental disorders were congenital cases (one in every 200 of vegetable and animal life was abnormal or a freak), and cases of toxic or infective origin; about one in four of general paralysis, which was infrequent, was associated with malaria. It was rare among the native population to find the highly evolutionized forms of insanity seen in Europeans, though melancholia, especially among the Jews, was frequent.

He believed that the effects of fear upon the mind, and chiefly the fear of failing to survive—i.e., the dread of failing to make both ends meet—were widespread and serious; but in the colonies most Europeans received fixed salaries, and fear from this should be less than that which caused anxiety at home. He disbelieved entirely in the sexual view of the origin of neurasthenia in our colonists. He regarded insanity as the product of two factors, stress and heredity, and no colonial servant should be selected who had a neuro-pathological family history; even at home in quiet surroundings these persons became erethistic and anxious. He considered that insufficient stress had been laid upon the dangerous effects of alcohol which Régis had specially pointed out many years ago.

Section of Electro-Therapeutics.

President—Sir HENRY GAUVAIN, M.D.

[*January* 20, 1928.]

DISCUSSION ON DIATHERMY IN RELATION TO CIRCULATORY DISTURBANCES, ESPECIALLY HIGH BLOOD-PRESSURE.

Professor S. RUSS (Middlesex Hospital).

SOME interesting measurements were published last year by d'Arsonval, who showed that the rate of generation of heat in saline and other liquids, viz., serum, urine and drinking water, was a very complicated function of the concentration of these liquids. Taking saline as a typical example of what happens, d'Arsonval showed that, if he kept the *current constant* through a solution, the rate of generation of heat increased with the dilution of the saline up to a certain point, and it then decreased very markedly.

The rise in the curve is what would be expected, for, in order to maintain the current, more power must be expended in the solution, and this should give a greater heat production; but the subsequent decline makes it clear that two processes are involved in the production of heat with H.F. currents.

As the salt solution is made more and more dilute, it gradually loses its electrolytic properties, but maintains its dielectric properties, and because of this, one process may become more and more important as the solution becomes diluted.

We may perhaps picture these two processes in this way.

(1) Linear oscillations of the ions caused by the H.F. impulses.

Taking an average value of 10^{-8} cm. for molecular diameters, and 10^{-6} for frequency of H.F. and the migration velocity for 10 volts per cm. as 0·02 mm. per sec., the distance travelled per impulse is approximately equal to 10^{-8} cm., i.e., a molecular diameter.

(2) Rotational oscillations of the neutral or uncharged molecules by the action of the field on electric doublets, giving rise to displacement currents.

Whereas (1) would be dependent on the local potential gradient, (2) would be dependent on local variations of the specific inductive capacity.

Some measurements, by Dr. L. H. Clark and myself, have shown that the generation of heat by these diathermy currents depends not only on the amount of energy consumed, but also on the frequency of the oscillations.

A horizontal tube was filled with a 10 per cent. solution of copper sulphate, and the rate of generation of heat in this solution was compared under different experimental conditions. It is hoped to publish the results of these measurements in some detail later, as it is thought that the differences in heat production caused by changes in the frequency of the oscillations are significant in medical work.

The details of one set of experiments may serve here to show the magnitude of the effect.

Frequency of Oscillation.	Joules per degree Centigrade.
50	506
2×10^5	580
$3{\cdot}3 \times 10^5$	698
$9{\cdot}4 \times 10^5$	757

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The general way in which big H.F. currents behave in the body is known very largely as a result of the observations of Cumberbatch. We may not, however, infer that a strict parallelism holds for strong and weak currents. In the medical—as contrasted with the surgical—uses, weaker currents are employed; it is, therefore, very important to have more information of the way in which the H.F. currents are distributed throughout the tissues and body fluids. The following observation, largely an accidental one, leads us to conclude that nerve tissue may provide an unusually good conducting path for H.F. currents, though the conclusion needs corroborative evidence.

On one occasion, a weak H.F. current, less than 0·2 ampere, was passed from hand to hand of one of the research members of the laboratory for less than half a minute; she said at once that she felt pins and needles in her right hand; that evening a large part of the surface of the arm was devoid of sensation. It should be noted here that owing to a cervical rib, a very small area on the inside of the forearm is always numb. The arm was examined by Dr. Peacock who kindly gives me the following information:—

"Diathermy current applied between two hands on Tuesday, January 10. That day she noticed numbness over the whole of the inner side of right forearm, especially on taking a hot bath. Thursday, on examination, there were no trophic changes in the right forearm or hand and no obvious difference in temperature between the two hands. Cutaneous sensation was tested and an area of anæsthesia on the inner and ventral aspect of the right forearm was mapped out by means of pin pricks. The anæsthetic patch was bordered by a narrow band of paræsthesia, the whole area corresponding accurately with the cutaneous distribution of the ventral fibres of the internal cutaneous nerve. There was no loss of sensation over the dorsal aspect of the forearm and the areas supplied by the ulnar were unaffected. The signs appear to be those of pressure on the ventral fibres of the internal cutaneous nerve which is probably looped over the tip of the *cervical rib*."

It seems justifiable to conclude that this effect can only have been produced by such a small current in so short a time if the bulk of it followed the nerve and caused dilatation of the vessels in the nerve itself and so increased the chance of pressure on it: a warning to the enthusiast that people subject to circulatory disturbances should not be experimented upon with diathermy currents. Two other people had exactly the same current for a much longer time and experienced hardly any sensation of warmth.

One matter I would like to mention is the necessity for voltage measurements in medical work with H.F. currents. The current which can safely be given depends on the applied voltage and the resonance conditions are doubtless very different in the various types of machines used. This means that a greater current can be tolerated from some machines than from others, so that a useful comparison between the power expended in the body in one case and another can only be made by measurements of voltage and current.

Dr. T. F. Cotton.

The value of diathermy in the treatment of cardio-vascular disorders depends upon the reaction of the blood-vessels of the body to heat. There is, I think, general agreement among electro-therapeutists that diathermy has no other special effect than the production of heat in the deep tissues of the body. We have in our possession—thanks to the recent investigations of Lewis and his co-workers—a series of accurately recorded observations on the response of the blood-vessels of the skin to stimulation from heat, which should prove of value in explaining the mechanism of the vascular response of the blood-vessels in the deeper tissues to thermal stimuli.

Lewis has advanced the view that the reaction of active dilatation of the arterioles, capillaries and venules to changes of temperature is a dual one; the direct influence of heat lessens the tone of the walls of these small blood-vessels, and a second indirect and predominating influence increases the concentration of vaso-dilator substances in the tissue spaces. This reactive hyperæmia, with its increased blood-flow is, as he states, a mechanism of fine adjustment governing the nutrition of the body tissues. Now the importance of this physiological conception of the regulation of blood-flow lies in the fact that these metabolites, which resemble histamine in their action, have a sustained effect, and are a normal constituent of the body.

When we come to apply this knowledge of the vascular reaction of the cutaneous vessels to a study of the effect of heat on the deep vessels of the body, we see that probably the small vessels in other parts of the body than the skin, react in a similar fashion. If this is the case, then the therapeutic value of diathermy in cardio-vascular disorders is related to these physiological facts. It follows from this explanation of the mechanism regulating the blood-flow, that when constrictor influences alter the peripheral circulation and cause pathological changes in the tissues, the release of this histamine-like body into the tissue-spaces causes the blood-vessels to dilate, the normal circulation is restored, and the tissues receive a better supply of blood. A reflex of central origin, by lessening the tone of the blood-vessels, also plays an important rôle in increasing the blood-flow to the tissues. Such a disturbance in the circulation we have in intermittent claudication. Theoretically, diathermy should be a valuable remedial agent in this condition. I have seen cases react favourably to this form of treatment; others have proved refractory, and have failed to show any improvement in their symptoms.

In acrocyanosis—another condition in which vaso-constrictor influences predominate—diathermy fails to relieve more than temporarily the venous stasis. Treatment spread over a few weeks is not sufficient to control the vasomotor influences which may be of central origin in these patients.

I approach with hesitation the subject of high blood-pressure and its treatment by diathermy. How much do we really know about this malady? Is there any common ground from which we can lay down laws governing its treatment? I believe, from the point of view of treatment, that we can divide patients with hypertension into two groups: (1) those with symptoms definitely related to the raised blood-pressure, and (2) those with symptoms due to other causes.

In the first group are many who feel more comfortable, and have fewer symptoms, when the systolic blood-pressure has been lowered 20 mm. or more. Some of these have obvious signs of arterial disease; in others, signs of structural damage cannot be recognized clinically. Diathermy lowers the blood-pressure in these patients and they are definitely benefited by this form of treatment.

In the second group, with symptoms due to other causes than high blood-pressure, are those with renal changes predominating, and with symptoms of renal insufficiency. A form of treatment which has as its main object the lowering of blood-pressure, is not rational for these patients, since it may lessen the functional efficiency of the kidneys. Diathermy fails to improve their condition. In the same group are patients with symptoms of myocardial exhaustion due to auricular fibrillation. When digitalis is given, there is a slowing of the ventricular rate which effects a change in the blood-pressure level. Diathermy is not likely to benefit these patients either.

Diathermy is a valuable therapeutic agent in the treatment of angina pectoris. I look upon angina as a symptom of myocardial exhaustion, and as often associated with disease of the coronary arteries. In patients with anginal pain and high blood-pressure, when the blood-pressure has been reduced to a normal level, it is probable

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that less work is required of the heart, and in consequence the circulation is more easily maintained. The improvement in the condition of some of these patients may be due to other causes than a simple lowering of blood-pressure by diathermy; the essential factor may be the increased blood-supply to the heart muscle through the coronary arteries from reactive hyperæmia caused by the raised temperature of the tissues. Here we have an explanation of the improvement which follows treatment of this kind in patients whose blood-pressure is within normal limits. When the blood-vessels from disease are refractory to heat, the blood-flow in the coronary vessels is not quickened by diathermy, and the pain persists unaffected by this form of treatment. A functional nutritional disturbance of toxic origin, a frequent cause of pain in secondary angina, is the most satisfactory type to treat by diathermy.

Dr. Justina Wilson.

Constitutional Arterial Hyperæmia.

Modern research has greatly altered our conceptions regarding this condition. The arterial pressure can be raised in two ways: firstly, by increasing the power of the myocardium, as in the case of aortic disease; secondly, by increasing the peripheral resistance in which case the arterioles, pre-capillaries, and capillaries bear the brunt of the pathological changes. These changes have been ascribed to a sclerotic thickening of the walls, hyaline changes in the intima, etc., but these are inconstant and often confined to limited regions, and workers have gradually been led to ascribe the primary change of a permanently raised blood-pressure to some functional condition of the arterial capillaries, all the other changes being secondary and due to wear and tear from the load placed on the cardio-vascular system.

Different parts of the cardio-vascular system react differently to this load, and the brunt falls on the renal, cerebral, and coronary vessels. How these changes come about is by no means clear. Toxins, undoubtedly, are a factor in the high blood-pressure of lead poisoning, nicotine, renal disease in toxic absorption from teeth and bowels, in many cases of prostatic enlargement and endocrine and climacteric conditions. In the majority of cases, however, no toxic factor is found, and if we exclude known toxic cases, we mostly have to do with patients with a constitutional tendency to hypertonia neither cardiac nor renal in origin. This forms the largest class both of hospital patients and of those seen in private practice. The late Sir Sidney Russell Wells and myself carried out some research on this question, and we found that in nearly 40 per cent. of the cases seen by us at the National Hospital for Diseases of the Heart, no cause could be found for the high blood-pressure. In the remaining 60 per cent. there were toxic factors due to arterio-sclerosis, syphilis, diseases of metabolism, scarlet fever, or nephritis.

Whether the primary condition be due to some disturbance of the autonomic nervous system, as Kylin has suggested, or to a decreased calcium content of the blood and a choleresterimæmia leading to a thickening of membranes on the surface of the smooth muscle of the arterioles, there is evidently some factor giving rise to a condition of constant spasm, the ultimate stage of which is autolysis and necrosis of the vessel walls.

Symptoms.—There may be none for years, the condition often being discovered accidentally. The first symptoms are related to the nervous system: morning malaise, anxiety, motor unrest, irritability. Later on, attacks of giddiness often amounting to vertigo. Others complain of "far away" feelings. These are really slight apoplectic seizures angio-spastic in character and may lead to cerebral hæmorrhage. Headache and decreased power of memory and concentration are common. Myocardial symptoms are precordial oppression and anginoid pains.

Though not in all cases, either of vasomotor or true angina pectoris, is there a high blood-pressure, many patients suffer from this and their attacks gradually become more frequent. Lastly, the signs of definite renal involvement show themselves.

The signs of myocardial damage are very definite: enlargement of the heart with a heaving apex-beat, the first sound at the apex is short, the second aortic sound ringing, this means true atheromatous change. Watching these cases over a period of years we were struck by the gradual appearance of atheromatous changes in the aorta: systolic bruits at apex and base, the latter conducted into the neck-vessels, pulsation in the suprasternal notch and a general broadening of the X-ray shadow.

Though it cannot be said that cerebral hæmorrhage is the inevitable end, yet in a period of seven years over 13 per cent. of these patients died of true cerebral hæmorrhage. The younger the patients are when symptoms first appear, the worse is the prognosis. By taking blood-pressure curves, on Kylin's lines, we divided up our cases into two groups: those with constantly high blood-pressure, and those with a labile or inconstant high pressure with marked variations, and we found the prognosis very much worse in the constant type. These curves—which can only be constructed in hospital or clinic as they necessitate taking the blood-pressures three or four times daily—enable one to establish a mean value for the blood-pressure which is more important for prognosis and therapy than is a record of the extreme heights reached.

Treatment.—Treatment of essential high blood-pressure can only be preventive and symptomatic. Before embarking on any mode of treatment, all unfavourable factors, toxins, complications, etc., should be dealt with, and the life regulated, and it is well to realize that there are spontaneous remissions in the earlier stages, and that the most important sign of definite improvement is a lowering of the mean value of the blood-pressure. In the primary stages of unstable blood-pressure it is difficult to gauge the success of treatment of these patients who go about, apparently quite well, with blood-pressures of from 180-200 systolic, while some have spontaneous remissions which are a fallacy.

In the stage of constantly high blood-pressure, symptoms become obtrusive and demand relief. Here there is still a prospect of improvement if we can get the mean value of the blood-pressure curve down by active and persevering treatment. The chief thing is rest, in the fresh air if possible, and early hours. Patients must from time to time be taken out of their usual environment, whether this be business, society or household cares.

Diet is of the greatest importance. A lacto-fruitarian-vegetarian diet is best, with cereals and salads, and with solids and fluids separated and avoidance of fermentation and further damage to the already burdened heart from an overloaded stomach and intestine. The diet must also stimulate peristalsis. Judicious massage may help here, and obesity must be dealt with. An occasional day or two in bed on either Karel's milk, or an exclusively fruit diet, greatly relieves both cardiac and cerebral symptoms.

Physiotherapy.—Hydro- and electro-therapy, diathermy and massage are indicated. Oxygenated baths have a certain effect in lowering blood-pressure; carbon dioxide baths are perhaps better if the myocardium is weak. Both must be followed by long periods of rest in a warmed bed. Wet packs (Priesnitz compresses) right round the whole trunk are helpful when there is flatulence, discomfort or insomnia; they are better than drugs for inducing sleep.

Mercury vapour baths are said to lower the blood-pressure. Personally, I have found them disappointing in chronic arterial hypertonia. Sinusoidal four-cell-baths often relieve symptoms.

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Diathermy.

We know that, by Joules' law, whenever the diathermy current passes through the body, deep heat is produced by the resistance of the tissues. The effect of such deep heating means a better flooding of the tissues with arterial serum, better resorption of pathological products, relief of pain and increased circulation.

Diathermy is extremely helpful in many cardiomuscular conditions.

Points in Application.

1.—As in the course of this treatment certain reactive phenomena may occur, it is best to have a day or two as an interval between treatments.

2.—Pain which lasts means that we either began the treatment too soon, or that the current was too strong. Thoracic and abdominal organs do best with medium currents. "The middle of the road" is the best guide in dosage.

3.—The correct placing of electrodes is of enormous importance.

4.—Diathermy treatments through the chest should never exceed fifteen minutes in duration.

Diathermy is indicated in angiospastic conditions, and the symptom complex of intermittent claudication. Here one can safely promise relief of pain and considerable improvement, also Raynaud's disease. In the early stages of gangrene whether diabetic, arterio-sclerotic or senile, it is possible to abort the gangrene by diathermy. But diathermy for the circulation should be combined with a very thorough treatment of the lymphatics. That is to say, the lightest possible skin massage to influence the superficial ducts, followed by a vigorous vibratory treatment of the deeper lying lymphatics, including the receptaculum chyli. This is often the keynote to success in the treatment of many circulatory conditions.

Even in true angina, the expression of coronary sclerosis, good may be done by diathermy, which decreases the severity and frequency of the attacks, though it does not alter the ultimate prognosis. I remember in Vienna a woman patient aged 58, a sufferer from essential hypertonia, with blood-pressure over 220, and latterly severe and frequent attacks of true angina. Diathermy was given through the chest. Symptoms, especially pain, were relieved. An hour after the sixth treatment she died. Post-mortem examination revealed the complete obstruction of a branch of the left coronary artery. Diathermy has been tried in heart block—both bundle block and arborization block—without success. The normal electrocardiogram is not much influenced by diathermy, nor has diathermy much effect on a sub-physiological curve with low or inverted T-waves.

In hypertonia, diathermy is good in relieving symptoms, but I have seldom found that it results in a definite constant lowering of the mean value of the blood-pressure curve, or a definite change from the constant to the varying type; but most of the cases I have handled have been serious ones in the secondary stage of constant high blood-pressure.

Dr. Agnes Savill

referring to the experiments of Bergonié and of Sloan, with high frequency currents on blood-pressure, said that the pressure was lowered, unaltered, or raised according to the degree of the response of the heart to the altered peripheral circulation. She described two cases treated by high voltage diathermy before the war, who had been watched over many years. One was a case of true angina in a man aged over 60, seen by the late Sir James Mackenzie, who had been much interested in the patient's description of the immense relief obtained by a course of auto-condensation, 350 ma. for fifteen minutes twice a week. The systolic blood-pressure had never

been lowered more than from five to eight points; the pulse-rate had scarcely altered, but to the examining finger had felt stronger and fuller. In the case of a neurotic young woman, the high frequency treatment had not altered the blood-pressure or the pulse-rate; descending spiral galvanism had raised the pressure four points. Another case of high pressure, in a man aged 67, had been watched for seven years, during which the patient had had two mild "strokes" affecting the legs. He had experienced benefit from two or three courses of high voltage diathermy, applied to the legs or abdomen; if over 450 ma. were given both systolic and diastolic readings shot up as much as ten degrees. With low voltage diathermy this patient had experienced more warmth, and the spasticity of the gait had improved more than it did with the high voltage apparatus; but the pressure readings had risen when more than 1 ampere for fifteen minutes was given.

Dr. ADOLF SCHOTT (Bad Nauheim).

Two cases of arterial hypertension with severe attacks of angina pectoris which were strikingly alike as to their clinical condition, illustrate the great individual difference in reaction on diathermy concerning the attacks. In the first case no method of treatment except diathermy influenced these attacks—even nitroglycerine was without any effect—diathermy, however, always checked the attacks in a short time. In the second case, nitroglycerine was the only preparation capable of checking the attacks, and in this case, diathermy, far from improving the condition, clearly tended to bring on the attacks. The blood-pressure remained unaffected in both cases. Cases in which diathermy might cause attacks of angina pectoris seemed to be rare, but the possibility should be borne in mind, therefore diathermy should only be used with great care in these cases.

Dr. E. P. CUMBERBATCH.

The distribution of the heat generated by high-frequency currents in gelatin conductors has recently been studied in my department by Dr. Simon. He found that the distribution was the same, whatever machine he used. One of the machines delivered current at a much higher voltage than the others, and the frequency of the currents was not the same for each machine, but showed considerable differences. His findings, which were of the *gross* distribution of the heat, were the same for the human body; the distribution was not influenced by the frequency or the voltage.

The phrase "treatment by high-frequency" should be discarded for that of "treatment by diathermy." The former expression was used when the action of high-frequency currents was not known. All the physiological and therapeutic effects of these currents can be explained as the result of heat. Heat can be measured and dosage rendered fairly accurate. If not due to heat, or if not wholly due to heat, to what are they due? How are we to measure or estimate dosage?

Dr. Cotton's description of the action of heat in vaso-dilatation is of great interest. The diathermy current directed through the skin, produces erythema which is, however, slight in proportion to the degree of heat. The skin can be heated to 114° F., yet the erythema is not intense. On the other hand an intense erythema can be produced by the application of brush discharges without the temperature rising higher than that of the blood. I think, therefore, that the increased blood-supply to the heated parts is due to acceleration of the flow, rather than to dilatation of the vessels. The acceleration may be due, perhaps, to diminution of the viscosity of the heated blood.

My experience of the effect of diathermy in cases of intermittent claudication confirms that of Dr. Cotton. Some cases are benefited; others are not. In cases of acrocyanosis I have found that diathermy had no therapeutic value. The cyanosis is replaced by a vivid erythema, but that soon disappears and the cyanosis returns.

Cases of high blood-pressure accompanying renal disease do not derive much benefit, in my experience, from general diathermy. In these cases the kidneys might be specially subjected to the treatment, for which some good results have been claimed.

All the cases of hyperpiesia that I have treated by general diathermy had already been treated on general lines. Most of them derived further benefit from general diathermy, and in a few, the blood-pressure was considerably reduced at the end of the treatment.

Cardiac diathermy is highly praised by Dr. Nagelschmidt for the treatment of angina pectoris. In this country good results have been described by Professor Hay and Dr. Dyson. It would be interesting to study the influence of cardiac diathermy on the electro-cardiographic record. Possibly it might alter the T-wave.

Dr. G. B. Batten

said he could not agree with the dogmatic statement that whatever good was done by diathermy was due to the heat produced. We had means of measuring the heat, but at present could not estimate the effects of the ionic and molecular movements caused by high frequency and diathermy currents. No doubt when means were devised for measuring them it would be found that heat was only one, even if the chief, of the means by which they reacted on the tissues. The experiments on the permeability of tissue by high frequency currents, described a few years ago by Dr. Phillips, of Belgium, showed that the current that could be passed increased enormously by increase of number of oscillations, and, he believed, out of all proportion to the heat produced. These experiments could not be neglected.

Mr. P. Bauwens

said he would suggest an explanation for the fact that better results were apparently obtained with high voltage currents in the treatment of high blood-pressure and its symptoms.

High voltage currents were apt to produce oxides of nitrogen (nitrous oxide and nitric oxide) where corona discharges or sparks occurred. These gases, being the anhydrides of the nitrite radical, might well, on being inhaled, behave in the same way as amyl nitrite, sodium nitrite, etc., and thus become a factor in the lowering of high blood-pressure and the relief of its attendant symptoms.

Section of Orthopædics.

President—Mr. W. Rowley Bristow, F.R.C.S.

[*April* 3, 1928.]

The Operative Treatment of Recent Fractures.

By Charles Scudder, M.D. (Boston, Mass., U.S.A.).

The treatment of fractures is the most important subject in surgery to-day, without exception, and for the following reasons:—

(1) In a case of fracture, the damage is not to the bone and surrounding parts only, but to the whole patient.

(2) It is necessary to decide at the time of the accident, if possible, upon the treatment appropriate to be carried through to the end of the case.

(3) Erratic, detached, unsatisfactory, traditional ideas of fracture-treatment are prevalent in our profession. The general practitioner is familiar with the accepted treatment of tuberculosis, of carcinoma, of the infections, of hernia, etc., but no comparable knowledge regarding the treatment of fractures is so widely disseminated.

(4) The initial treatment of a fracture may stupendously affect subsequent events—a relatively simple task may be rendered complex, difficult, or well-nigh impossible.

(5) A simple effective primary treatment may be comprehended and successfully applied by every general practitioner of medicine and surgery.

(6) Ambition on the part of the surgeon to secure better results is stimulated by the demand of the patient, by the interest of the employer, by the solicitude of the insurer of the employer, and by the demand for a decent standard of living. All these factors tend to raise the standard to be reached in treating a fracture and render unsatisfactory the standard of even five years ago.

(7) The type of fracture has changed decidedly in recent years.

There are more complicated joint involvements, more multiple fractures. There is greater damage to the bone and to the soft parts in civil fractures. The problems presented are often greatly involved; fractures are now more difficult to treat. Better training and very special general surgical judgment are required.

We all agree that many cases of non-union, mal-union with symptoms, and compound fractures, require operation. These cases are excluded from our present discussion.

The Great War, industrial developments, complicated machinery, Workmen's Compensation Acts, the free choice of the injured workman in selecting his doctor, the increased use of motor vehicles—all are tremendous stimuli to the surgery of fractures.

With the advent of anæsthesia, asepsis and antisepsis, and the Röntgen ray, remarkable strides were made toward the perfection of many branches of surgery. Diseases of various organs often killed the individual, hence the best minds in the profession studied the surgery of pathological processes.

Fractures of the skeleton rarely killed—they only incapacitated; little attention, therefore, was bestowed on them. When it was recognized that the mortality of open fractures was 80 to 90 per cent., the work of Pasteur and Lister showed the way to diminishing the terrible mortality. To-day, an ordinary civil compound

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fracture is safely treated. The operative treatment of certain war fractures confirmed for ever the principles evolved by Lord Lister, and on these principles the operative treatment of fractures rests.

I can recall watching with awe, thirty-five years ago, the late Dr. John Collins Warren, at the Massachusetts General Hospital, score and slash a closed fracture of both bones of the leg for impending gangrene due to tension from hæmorrhage following injury of important vessels below the knee. The anterior and posterior tibial pulses were absent; the toes and foot were blue and cold. By these incisions Warren rendered a closed fracture open. The outcome of this logical and bold step was watched with apprehension. The patient recovered with a sound leg.

Compare this episode with the assurance accompanying a premeditated incision down to the bone to-day. The change in attitude towards such a procedure is almost unbelievable. To-day, timidity has given place to boldness; emergency measures, to deliberate operations. The operative treatment of fractures has reached its present important position comparatively recently.

The present serious discussion of the operative treatment of fractures coincides with the present technical perfection of pathological surgery. It coincides also with the entry of surgery into preventive medicine.

It is as if the surgery of pathological conditions had been improving and developing up to and for this great event.

The aseptic régime of general surgery makes possible the treatment of fractures by operative methods, but rigid conformity to established aseptic ritual is essential to success and is postulated in this argument.

We must strive more energetically in fracture-treatment for *anatomical perfection*, which means a nearer approach to functional perfection and the prevention of conditions caused by inaccurate replacements. For example, if the fracture of the external condyle of the humerus were always exactly replaced by operation, if necessary, there would be fewer late ulnar nerve paralyses.

To point to cases of fracture of the shaft of the femur in adults—fractures not involving a joint treated by manipulation, or by skin traction, with poor anatomical, but with good functional results—as justifying the expectant, non-operative treatment, is not sound surgery. Rather is such an experience an argument for better non-operative reposition, or for early operative reposition of the fragments. In addition, the result would probably be secured in less time and with less discomfort. Rapid repair is consistent with absolute rigidity.

There are to-day positive indications for the attempt to secure good form, and only a negative one for allowing poor form. The calamities resulting from misuse of operative methods are arguments not against proper surgery, but in favour of attempting ideal surgery.

It is dangerous to advocate incomplete reduction of a fracture in an adult, and thus to sacrifice the possibilities of operative procedure, or of the more careful use of non-operative treatment. Surgical compromise stunts progress and may cause surgical cynicism, but though we seek the ideal, we are justified in an occasional intelligent compromise.

The desirability of anatomical reposition more and more often demands direct operative approach.

Legal liability for failure to use X-rays as an aid to diagnosis and treatment implies increased responsibility and a greater necessity for accurate adjustment of fracture fragments. To say that we want, not X-ray results, but good function, is unfair to the legitimate use of the X-ray. X-rays call for the open reduction of certain fractures.

A more precise and comprehensive conception of bone exists to-day than ever before; it is no longer regarded as dry, inert, stable matter forming the passive framework of the body. It is a flexible, living, changing tissue—an active organ, intimately associated with the vital processes. The bony skeleton is a protection, a support, a means of locomotion related to certain physiological processes, a reservoir of calcium. Bone tissue is sensitive to all extraneous influences. The reaction of bone to trauma, to fracture, is recognized as a complicated series of events—the processes of repair; that bone tissue must be gently handled during manipulation and at operation is now appreciated.

This view emphasizes the importance of immediate, exact reposition of fracture fragments, so that the reparative process is least hindered, and ideal union accelerated. This necessitates the finest technique known in surgery. Successful technique in bone surgery is unexcelled in delicacy and precision even by the gentleness recognized as necessary in handling cerebral and abdominal tissues. This conception of bone cautions us against the blind manipulative traumata of repeated attempts at reduction, and points to the operative treatment as the ideal in many cases of fracture.

A fracture is an injury of a complicated and intelligent mechanism. The will is involved in every fracture; it influences the recovery of function. The recovery of normal function of the whole organism is at stake—not merely the healing of a broken bone.

The less function is interfered with, the earlier the part is restored to normal. The earlier the will of the patient coöperates in securing this return of function, the more rapid and complete is the recovery.

Following a fracture the parts may be moved as early as possible without disarranging the replaced fracture fragments. There can be no rigid rule as to the time of union of a fracture. Further experiment and careful clinical observation upon the time of union of uncomplicated and complicated fractures are needed.

Clinical experimental work upon the proper application of massage is demonstrating a far earlier safe union of fragments than seemed possible a few years ago. The old ideas of the necessity of prolonged immobilization unfortunately still influence treatment. We are all coming to allow the fracture to interfere as little as possible with the function of the part, including the muscles and joints.

The principles of movement following injury, established by Lucas Championnière, and followed and taught by Mennell in England, are now recognized by all of us as sound, and now occupy a permanent place in fracture treatment; their acceptance has profoundly influenced fracture treatment. We should secure perfect return of function at the earliest possible moment after injury, and this demands the treatment of many fractures by operation.

Absolute fixation by operation secures a minimum of callus (i.e., the minimum compatible with sound and secure repair), and permits the earliest movement of joints without disturbing the reposed fragments.

Extra-uterine pregnancy used to be treated solely by rest and non-operative measures. Then came the saving of more lives by operative procedures. We all know of the catastrophes following treatment by expectant, watchful methods, in appendicitis, and we recall the stormy discussions over operation; finally, we recognize to-day early operative treatment in most instances as the treatment of choice, with a resulting diminished mortality and a quicker return of function.

Up to ten years ago operations for possible spinal-cord tumour were not done until there were present the classical clinical pictures of spastic paraplegia, stiffness of the legs, and a definite level of anæsthesia—a nearly hopeless condition.

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Mistaken diagnoses were made, and consequently negative exploratory operations resulted in many cases.

With present diagnostic methods, especially designed to show block in the spinal fluid, pathway operations are done with the certainty that an obstruction exists.

By comparison with the progress of treatment in other surgical conditions, operative treatment in fractures is seen to be logical, and it stands on the firm basis of established asepsis.

As our ideas of the nature of bone tissue develop the operative treatment appears the least harmful—a gentle method of handling this sensitive tissue; and further, the coöperation of the patient's will power may early be secured if the direct treatment of a fracture is followed.

Indications for Operation.

What is the direct indication for the operative treatment of recent fractures?

We must first assume that:

The highest degree of safety to the patient will obtain.

The surgeon and his assistants are skilled in the treatment.

The surgeon possesses ability greater than that legally required.

The surgeon has available the necessary instruments and apparatus.

The use of the form of anæsthesia suitable to the case will be skilful.

The final and exact procedure chosen in the operative treatment of a given case is appropriate.

The pre-operative and post-operative care is adequate.

By this treatment the involved or contiguous joints are moved as early as possible.

The important indication is that by the open operation an earlier return to a more nearly normal function will be secured than by any other treatment.

The operative treatment of fractures is no longer a last resort, it now occupies an established place in surgery; it is in many instances an initial method of choice. The recognition of this is a great step forward. The harm done by delay and by obsolete methods of bloodless surgery will be reduced to a minimum.

It is to-day less a question of non-operative versus operative treatment than it is a matter of selection of those cases which are suitable for each method.

Fifteen years ago, in his Presidential address at the Liverpool Medical Institution, on "The Present Position of the Treatment of Fractures," Sir Robert Jones said: "Must we prepare ourselves to admit that primary operation is to become the recognized routine? Rather, there are two questions. Can we improve non-operative technique? Can we lay down any laws to guide us when we ought to operate at once?"

In discussing Arbuthnot Lane's first paper at Atlantic City in 1909, I said: "The time is not ripe for the general employment of the operative treatment of fractures, but the important facts disclosed by Mr. Lane's study of the bad results of fracture treatment should be a strong stimulus to improving the non-operative methods which are rarely honestly applied."

Mr. Lane did demonstrate the innocuousness at times of huge masses of non-absorbable metal within human tissues.

He deserves credit for his emphasis of the crippling results of badly treated fractures and the need for an improvement in the handling of fractures. This was a distinct and sorely needed advance in fracture-treatment.

Though the conditions enumerated demand the treatment of certain fractures by operation, this does not mean that every fracture should be operated upon. The honest and intelligent use of non-operative methods suffices for most fractures.

Great advance in fracture treatment will inevitably be along operative lines; this advance should be safeguarded and limited to skilled surgeons in suitably equipped hospitals, and should be further safeguarded by the control of a group of surgeons in the particular hospital.

There never can be standardized methods of treating fractures. There are many methods of carrying out effectively the principles underlying fracture-treatment.

The younger generation of surgeons must acquire the facts and technical details of both methods of treatment established in the past few years, so that they may not repeat needlessly experimental work upon the patient and so handicap recovery.

A few details will make my meaning clear:—

The limitations of skin traction in adult and child; the conditions governing the efficacy and limitations of skeletal traction; the ineffectiveness of intrinsic Thomas's splint-traction in certain fractures of the shaft of the femur; the great value of the Thomas's splint for transportation, for suspension-traction and immobilization purposes; the desirability of suspending a broken limb; the early recognition of fractures difficult to handle; the value of massage in the reduction and subsequent treatment of a fracture; the safe time-limit for trial of non-operative treatment; the desirability of an immediate reduction; all improvements in operative technical details; the preservation of the normal contour of the diaphysis of the long bones; the improved operative approaches to fracture fields; that some non-operative methods have been tried under certain conditions and found generally wanting; that every suspected and actual fracture should be radiographed before and after treatment is begun; that any belittling comment on a fellow practitioner's treatment of a fracture may mean a medico-legal trial and an unjustly damaged reputation.

Those of us responsible for the care of fractures and interested in seeing better treatment cannot too often stress the importance of these details.

Mr. Hey Groves recently emphasized in a very telling fashion: (*a*) the need for a critical examination of the fracture during the few days succeeding its apparent reduction and satisfactory immobilization; (*b*) the danger of inefficient steel plating operations; (*c*) the danger attending indiscriminate plating of compound fractures.

What of the proper treatment of the three or four hundred thousand fractures occurring in the United States and Canada in a single year? What of the thousands of fractures occurring in Great Britain in a single year?

Most of these are cared for in the first instance, and always will be, by the general practitioner.

There are ideal simple methods of treating a fracture when the injury is first received. Every local physician can and should qualify in the first-aid treatment. Such adequate immediate treatment will prevent many subsequent difficulties. The time to be worried about the outcome of treatment is within the first two hours after the injury. Then if the local physician will always secure the advice of a consultant and if they both recognize at the outset that the case is difficult, and refer it to some one competent to care for it, serious troubles will be avoided.

If experimental advances in fracture surgery are left to those especially qualified in safeguarded hospital centres, the future of fracture treatment will become a credit to general surgery.

A surgeon who operates on recent closed uncomplicated fractures should possess the following qualifications: An adequate knowledge of the patient, physically, mentally, socially, industrially; an exact knowledge of all the conditions bearing on the case; perfect instrumental, mechanical and physical equipment, including access to X-ray apparatus; an accustomed, proved operative technique, delicately balanced; an understanding of the sensitive nature of bone tissue, its reaction to injury, and the conditions influencing the process of repair; a practical experience of the

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successful treatment of certain fractures by modern non-operative methods; a knowledge of the various tried and accepted operative procedures for the approaches to and the immobilization of fractures; an open mind in selecting the method best adapted to the case in hand.

A surgeon with these qualifications is capable of safely treating fractures by operation. A surgeon not having these qualifications should not employ the operative method in treating fractures.

If one is master of the general principles and technical details of both external and internal fixation, is informed as to the results of such treatment, has a complete knowledge of the proper use of massage and active movement and can secure the patient's complete coöperation, one's vision of the surgical treatment applicable to a given case of fracture of bone will be so broad, one's selection of the treatment will be so wise, and one's execution of the treatment chosen such that the result both functionally and anatomically will be the best obtainable for that particular individual under the existing conditions.

I have attempted to state the bases on which the operative treatment of fractures rests. These bases are solid, sound and enduring. I have indicated the absolute necessity for the limitation of the operative treatment to those competent and equipped for it.

With our present limited knowledge of the results of ideally conducted non-operative methods and the results of ideally conducted operative methods, it is impossible to state a final opinion of the applicability of one method of treatment to a certain fracture. Our opinions will necessarily vary.

Under present conditions fractures fall into three groups: (1) Those never operated upon; (2) those always operated upon; and (3) those in which operation must be looked upon as of doubtful applicability.

In the first group will be found Colles' fracture, most fractures of the clavicle, many fractures in childhood or the adolescent period, and many birth-fractures.

In the second group, those usually operated upon, will be: Fracture of the great tuberosity of the humerus with displacement; fracture of the surgical neck of the humerus with dislocation; fracture of the olecranon with separation of the small fragment; fracture of the head or neck of the radius with such displacement of the small proximal fragment as would, without operation, produce limitation of pronation and supination and possibly limitation of elbow flexion and extension; fracture of the shaft of the radius with displacement toward the ulna; separated epicondyles of the humerus; displaced condyles of the humerus, not held by the acutely-flexed position or when the acutely-flexed position is contra-indicated.

Certain adult elbow-joint fractures: Irreducible fracture of the shaft of the femur, at any level; a displaced femoral condyle; fracture of the patella with displacement; certain spiral or oblique fractures of the tibia and fibula; fractures of the os calcis in which the line of fracture enters the astragalo-calcaneal joint; fractures about the ankle-joint difficult to hold; certain metacarpal and metatarsal fractures.

Rarely would I operate, at present, upon a recent fracture of the neck of the femur.

In the third group, those in which operation is of doubtful applicability, are:—

Fractures of the spine with immediate symptoms of transverse lesion of the cord; fractures of the humeral shaft above the middle; fractures of both bones of the forearm.

It must always be borne in mind that the proper use of skeletal traction upon the condyles of the femur, or the tibial crest, or the malleoli, or through the os calcis, may diminish the necessity for the operative treatment by direct incision as an initial method of choice.

Almost every fracture of the neck of the femur is within the capsule. In our clinic at home, the treatment for fracture through the middle of the adult neck—the one which is difficult—and the one near the head, would be the use of traction under a general anæsthetic, i.e., traction, internal rotation, abduction and hyperextension. Hyperextension of the lower extremity is important as securing tension on the anterior portion of the capsule, so helping to "snug" things together. That method would be used in the ordinary case: 60 per cent. of the cases coming to Massachusetts Hospital have secured bony union by that method, and all were X-rayed before, immediately after, and some time after. The 40 per cent. who have not secured bony union constitute a stimulating problem. Is there any method by which we can determine beforehand that union will not occur in a given case? I think not. But we may know, by a careful study of the skiagram, that the neck is being absorbed and that union will not take place without waiting for three months, whilst the leg is being kept abducted. In our clinic we are inclining more and more to the reconstruction operation rather than the pegging operation, because the former is known to give a good, stable, serviceable lower extremity, and the latter, under such conditions, does not give such good results. So, other things being equal, that would be the second choice.

Dr. Cotton, of Boston, believes in artificial impaction of a fracture, and he is using that instead of abduction treatment; he puts his stockinged foot into the patient's groin on the injured side (the patient being under an anæsthetic), takes hold of the lower extremity and makes traction until the legs are of equal length. Then, inverting the lower extremity, much as one would in putting up a fracture, without artificial impaction, and having the pelvis supported, he puts a double piece of saddler's felt over the great trochanter, and with a wooden mallet having a handle a foot long he hits the trochanter and impacts the neck in this corrected position. The skiagram proves that he accomplishes it. He believes that instead of simply placing the fragments in apposition in the abducted position, which gives union in 60 per cent. of cases, this impaction is better. Cammell, of Tennessee, has been following Cotton's practice, and they both have series of cases which are being studied. It is a question whether this procedure will prove effective in that 40 per cent. in which union is not obtained by the other method.

Nailing or pegging with beef-bone, or with an autogenous peg, still remains a treatment of choice. I have seen Delbet's cases in Paris. He pegs practically all the adult cases at the neck of the femur, and I understand that in about 50 per cent. he secured union. In another clinic in Paris they have developed a surprising piece of apparatus by which, checking it up with X-rays, locating the femoral head, and adjusting the apparatus with the patient on the table, they place a bone-graft in the neck of the femur exactly correct every time. They do not refresh the fractured surfaces, which is not altogether right in the non-union cases.

After all has been read and said, I believe in the non-operative treatment of fractures, but I believe also in the operative which has come to be a necessary part of fracture treatment. It is logical, it is sane, and it is established, errors and difficulties aside.

Discussion.—Sir ROBERT JONES said he was pleased to find that Dr. Scudder, though a strong advocate of the purely operative method, also urged perseverance in the non-operative method. He (the speaker) would not wish to be a member of any particular "school," because the truth was not in any one school, or, perhaps, in all the schools put together. He thought that much depended on the particular surgeon. The surgeon should do what he was able to do well. Some time ago he had had Championnière in his room, and had shown him one or two fractures with deformity, and asked how he would treat a Colles. Championnière's

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reply was that he would not trouble about rectification; he would "massage and massage." Asked to show how he would do this, he beautifully and skilfully carried out massage for an hour and a quarter. There must be a reason for such a conservative attitude, as he (the speaker) had long maintained. A certain school, on the other hand, went "the whole hog," and the speaker discounted their operations because of what he saw. At the period when these operations had run riot, he had had painful experiences in his clinics. He had seen babies who had been operated upon; he had, in fact, removed a plate from a child aged 6 months! Frequently plates were removed from cases which had been treated two or three years before, but these plates had now begun to give trouble; that was not uncommon in cases of fractured patella. He had also had cases in which Colles' fractures had been operated upon. One surgeon—for whom he had great respect—had said in a debate that he looked upon Colles' fracture as one of the most suitable kinds of case for operation. It was necessary at that time to go almost to the other extreme in order to counteract this tendency.

He wished to draw particular attention to fractures of the femur. Dr. Scudder had said one had to operate on these to get what he termed "perfect anatomical reposition." Perhaps that was where he (Sir Robert) differed from Dr. Scudder. If one said: "I would like this bone to be absolutely together," well and good, but much depended on which was the bad type and which the good type of union. In the femur, end-to-end apposition was good, but if there was even slight deflection, it was bad. A slightly overlapping fracture, if in good alignment, was a better result than an end-to-end apposition which had been allowed to cant on one side. He had seen cases plated in which that point had not been remembered.

During the War, many people at first were satisfied if the femur, after treatment for fracture, showed only an inch of shortening. But when cases came to be treated in hospitals by men who understood the treatment (such as Sinclair, Pearson of South Africa, and others), though at one time there were in the hospitals 500 cases of fracture of the femur, many of very bad type, the average shortening did not exceed half an inch. Since then surgeons had very rarely set out to plate fractures. Sometimes the surgeon would say he had bared the bone and made a pull on the lower end, and yet could not make up the shortening. It was important to remember that in the femur the good results were obtained by gradual extension rather than by immediate reduction. That was the great difference between the direct operative attack and the attack by other methods.

He agreed that often spiral fracture of the tibia needed operation; but the case which required the operative attack was generally that in which there was not only a spiral fracture, but also shortening. Skiagrams might show the bone cut across and one end rolling over the other; the appearance was alarming, but this was not a bad fracture; all one had to do was to rotate the ankle, and then the bones came into perfect apposition.

Forearm fracture was sometimes difficult to deal with, especially when the radius came into contact with the ulna. But, here again, operation was not so necessary as one might believe. He (Sir Robert) had looked after thousands of cases and had only been obliged to operate in ten or twelve. He was, however, very careful in the matter of reduction. When cases came in with limited pronation and supination of the forearm, the arms generally lay in pronation more than in supination; very rarely did he see an arm completely supinated which showed any marked difficulty in pronation. He therefore taught the fixing of the fractured forearm in almost full supination.

With regard to the "head-of-the-radius" cases, when one saw a case in which that piece of bone had been knocked off, it was always best to remove it, as much trouble might occur afterwards, and removal of the bone after the third week was not as successful, because so many adhesions had formed.

In cases of fracture at the neck of the radius he would always avoid cutting down on the neck of the bone; there was little opportunity of fixation, and if it were kept well supinated the result was usually good.

One of the few fractures in regard to which he would operate was that of the scaphoid. The main trouble there was dorsiflexion. Sometimes there was limitation on flexion of the wrist also. The first thing he did was to increase the deformity, putting as great pressure as he could on the scaphoid and semilunar, trying to force it back as much as possible. If there were an easy movement of dorsiflexion he considered that the case would do well; otherwise he thought it wise to cut down at once and remove the obstructing bone. It was

better to remove the scaphoid immediately one knew one could not get full dorsiflexion than to do an operation weeks or months later. In the latter event the operation was usually only a partial success.

With regard to the patella. In the case of a fractured patella in a young man he would always operate by stitching, rather than by inserting pegs or plates or wire. This operation would be only performed on athletic people who wished to make the fullest use of the knee. It would be against his judgment to do it in the case of old and feeble patients.

In an interesting paper Mr. Hey Groves had drawn attention to the acute flexion in different fractures of the elbow-joint. We did not push the arm as far as possible. There was a distinct method of reduction, which was not that of taking the elbow and flexing it. [Demonstration.] If there were a swelling at the time of the fracture this method should be employed very carefully.

Litigation trouble, with regard to fractures, generally occurred because of some unforeseen happening, such as ischæmic paralysis, which had not been recognized, and concerning which the patient had not been forewarned. Whatever means might be employed, one was apt to see irreparable accidents to the soft tissues at the time of the accident or fracture. Foot-drop might occur, for instance, following fracture of the upper part of the fibula, or ulnar paralysis following displaced internal condyle of the humerus. If the doctor had not noticed before his manipulation that paralysis had occurred, his position was very awkward.

It was a common thing to plate Pott's fracture, but Pott's fracture was one of the easiest to manipulate if seen early, and the results were generally good.

If one watched the entrance of patients in these cases of fracture one would find indications of what was to be guarded against. A patient, e.g., came with fracture of the femur, and on inspection it was found to be hyperextended, or bowed on the outer side, indicating that there had been sagging just above the joint. This could be avoided by mechanical means.

In cases of fractures of the tibia, some plated, some unplated, the patients had usually a deflection to the outer side of the lower end of the tibia, i.e., the normal curve of the tibia might be straightened, and, owing to the flat-foot which followed the injury, there might be a difficulty at the knee-joint.

In the shoulder there was another interesting condition; one had to be careful about a shoulder fracture; the scapula must be fixed while the abduction was being made, otherwise the result would be bad. If one decided that there was a fair chance of putting a fracture into a good position by not operating, it was one's duty not to operate. If, on the other hand, the skilful operator said: "This is the only way I can treat it," let him so treat it. But, though any school might maintain that a certain course was best, they should refrain from saying that other methods were unjustifiable.

Mr. HEY GROVES said it was generally agreed that the broad indication for operative treatment of recent fractures was the failure of closed methods to produce satisfactory reduction of the fragments. But this formula still left open two points, on which there existed great diversity of opinion and practice. The first was:

What amount of displacement was consistent with the ideal of "satisfactory reduction"? The second was: In what cases was it useless to attempt closed methods?

With regard to the first point he considered that the necessity for perfect anatomical reduction had been overstressed by the advocates of open operation. Provided that the limb was restored to full length, and that the general alignment was correct with actual contact of the fragments, some degree of lateral displacement could be ignored. In such cases firm union and functional recovery would be quicker by closed than by open methods. In his opinion it was abundantly proved that open operation, especially plating, actually delayed bony union, even if no septic or irritative complication occurred. This was probably due to the devascularization of the bone ends, necessitated by wide exposure and the separation of the soft parts from the bones. In a considerable proportion of cases, plating was the cause of the non-union, even when perfect aseptic healing of the soft parts had occurred. He would lay special stress on the bad—and even disastrous—results which followed the plating of open fractures. An open fracture, with the displaced bone ends exposed in the wound, seemed to be an irresistible temptation to many surgeons. Then followed, usually, either sepsis—trivial or severe—devitalization of the bone ends, removal of the plate and a final

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condition of non-union, with perhaps necrosis, which might at last necessitate amputation. It was probable that after one such experience the surgeon would not repeat the adventure, but it was quite unjustifiable that any surgeon should need even one disaster of this kind to teach him wisdom.

In regard to the second point, namely, the cases in which it was a waste of time to use closed methods, he would suggest the following: fractures of the patella and olecranon, with wide separation of the fragments; fractures of the articular ends of the bone in which manipulation failed to secure reduction, especially when these fractures were associated with dislocation; fractures of the radius, or of the radius and ulna, with ulnar deviation of the distal radial fragment; fractures of the true neck of the femur in vigorous and healthy patients.

The actual technique of the open operation presented a very wide field for discussion, and he was only able to touch upon two points. It was generally considered that the use of unabsorbable metal structures was better avoided, and he entirely agreed with this opinion. Though admitting that in certain cases, efficiency of union fully justified the use of these materials.

It was true that encirclement of the patella by a wire gave very good results, but in some cases there was a resultant tissue-reaction in and around the knee-cap, which caused limitation of movement. This could be avoided by using a broad strip of fascia lata in place of the wire.

In treating fractures of the long bones, particularly those of the humerus, the radius and the tibia, by open operation, it was usually possible to bring about perfect reduction and to maintain it without the use of any permanent bone-sutures.

The simplest method of manipulative reduction was to force the bones into apposition, with the guidance of the fluorescent screen. But in many cases the interposition of soft parts made this impossible and open exposure of the bone ends was necessary. It was then, possible, as a rule, to impact the ends so that they would remain in place. He had found that this simple type of operation tended to accelerate callus-formation. This type of impaction, under the guidance of open exposure, was particularly useful in the upper end of the humerus, or in the shafts of the radius and ulna.

There were cases, however, particularly those of oblique or spiral fractures of the tibia, in which, after the bones had been replaced in accurate apposition, they tended to become immediately displaced when they were released. In these circumstances it was usually possible to obtain adequate fixation of the bones without burying metal. This was done by partly transfixing each bone fragment by means of a bradawl. The two bradawls were arranged in different axes, and were held in position while the wound was closed and the limb was encased in plaster. The plaster would encircle the shafts of the bradawl, and when it had set, the broken bones were kept securely in place. The bradawls were removed a day or two later without disturbance of the casing.

Mr. H. A. T. FAIRBANK said he would confine his remarks to the application of the general principles (so ably dealt with by Dr. Scudder and the subsequent speakers) to special cases, since this point constituted the chief difficulty of the student and the inexperienced practitioner who was often in doubt, after study of the radiograms in a given case, whether a certain displacement demanded correction or could safely be disregarded. The surgeon should always keep in mind the great difference between operating in the first week and operating after a lapse of, say, six weeks. This was particularly important with fractures involving joint surfaces. In these, absolute perfection in the anatomical result of operation was necessary, but might be impossible unless the fracture was quite recent. Cases had to be left uncorrected only because they had not been seen early enough. The day of election for operation was the fourth; unnecessary delay only meant the sacrifice of more reparative cells and osteoblasts in clearing the fragments.

The following points were important:—

FRACTURES OF THE HUMERUS.—*Shaft: The* indication, almost the *only* indication, for operation was damage to the musculo-spiral nerve. *Internal or External Condyle:* If displaced to more than a minute degree, operate. In external condylar fracture, by no means rare in children, the fragment might be rotated more than 90°, a condition often not appreciated. *Supra-condylar:* If the usual displacement was complete and could not be reduced by

manipulation, open operation was advisable, provided it could be done within a week of the injury. If six weeks or more had elapsed, gross displacement was far better left alone. A nerve-lesion should be regarded as one factor in favour of open operation. When the displacement was slight or had been already reduced, nerve lesion, *per se*, was not an indication for immediate operation.

FRACTURES OF HEAD AND NECK OF THE RADIUS.—When in doubt, operate. If the head was completely loose, remove it, but if the periosteum was intact to a large extent, e.g., in separation of the epiphysis with displacement, do not remove it but correct the deformity. If the head is cracked, remove any displaced fragments. A gap or depression was preferable to a projection on a joint surface.

FRACTURES OF THE SHAFTS OF THE RADIUS AND ULNA.—If overlapping, operate in most cases. If transverse, accurate replacement would suffice; if oblique, plate one or both bones.

COLLES AND SEPARATED EPIPHYSIS.—Only required open operation when united in bad position and could not be refractured, e.g., after four weeks or more. Separated epiphysis demanded the same treatment, but this was not so imperative for a relatively small degree of tilting. Correction by manipulation was more difficult, but union was more rapid, and operation might be required earlier in these cases.

The displacement in a Colles or separated epiphysis that really mattered was backward tilting. Moderate displacement backwards with no tilting was unimportant from the functional point of view.

FEMUR.—*In fractures either of the neck or of the shaft*, operation should be required only in exceptional cases, if treatment was properly carried out. Plating of the shaft of the femur was advisable, without attempting conservative measures, when the soft parts below the knee were damaged or there was a second fracture in the limb. In the lower third, admittedly more difficult to treat, operation was more often advisable. This was particularly true of separation of the lower epiphysis. Correction of displacement, if considerable, in these cases appeared to be peculiarly difficult by manipulation alone.

Condyles.—Operation was imperative if the displacement was anything but extremely slight.

TIBIAL TUBEROSITIES.—These were extremely difficult to deal with by open operation, since the fractured surfaces could not be cleared without further displacement or damage to the joint, and good clamp fixation could not be maintained while screws were inserted. Great care was necessary before deciding on operation. If fair accuracy could be achieved by manipulation the fracture was best left alone.

SHAFTS OF TIBIA AND FIBULA.—Most favourable cases for operation. Want of apposition of fragments was alone sufficient reason for operation, while overlap exceeding a quarter of an inch in oblique fractures made it imperative, in the absence of any contra-indication.

In transverse fractures of both bones with complete disengagement of fragments, always operate however slight the overlap might be at first. The *oblique* fractures, if requiring operation at all, should be plated; the *transverse* usually require no internal fixation. Though he (the speaker) had almost invariably used a plate and screws in these cases, he had been impressed by the extremely close apposition of the fragments when Parham bands were used.

POTT'S FRACTURE.—More than any other joint, the ankle requires absolute accuracy and perfect fit. The lower the oblique fracture in the fibula the more likely the need for operation. In the young active adult comparatively slight displacement would give a considerable percentage of imperfect results unless operated upon. In those of the third degree operation was much more often advisable and should be resorted to without hesitation even in patients somewhat past middle life. In cases of all sorts the slightest outward tilt of the astragalus demanded operation if it could not be corrected otherwise. Backward displacement of the foot must, of course, have been entirely overcome.

[The points referred to were illustrated by a number of slides.]

Mr. W. A. COCHRANE said that, surrounded as one was, in the case of fractures, by many impressions and few facts as to end-results of treatment, final judgments as to the relative merits of different forms of fracture treatment were impossible until accurate records of

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comparable cases treated by different methods were available. In this respect the Fracture Committee of the American Medical Association was accomplishing a great deal. It was encouraging the establishment of fracture services in hospitals. This meant the provision of adequate equipment and a trained staff, who, by frequent conferences and the following up of the cases, would gradually get at the facts. It would be a great boon if special fracture services were established all over this country, as they improved the training of students and surgeons in a remarkable degree. Until there was a radical alteration in the system in this country the good resolutions of experts as to standardization of methods would be of little more than academic value. The method of treatment adopted in any one case was governed by the capabilities of the person into whose hands it came. It was agreed, however, that certain fractures usually required operation. In the first group were intra-articular and juxta-articular fractures, such as fracture of the patella, olecranon, separation of the capital epiphysis of the femur and fractures into the elbow, knee, or ankle. The restoration of the normal alignment of a joint was essential to good function, especially in the lower extremity. In many cases this must be accomplished by operative replacement of a separated fragment. In other cases the offending fragment could not be replaced and should be removed. In very severe fractures into a joint, such as the knee or foot, arthrodesis was best. Thus, one of three methods had to be chosen. With regard to the first and second procedures, however, it must be emphasized that early active movement was just as important as the operation to restore function. This point was too often neglected.

In fractures of the shafts of the long bones, operation should be performed only in selected cases, and only after preliminary traction had been tried. It was important to bring the limb down to length before operating. The majority of cases could be efficiently treated by non-operative methods. In the case of the lower extremity the good effects of efficient skeletal traction and the use of lateral pressure-pads had produced the most striking improvement in results. The strongest rival of operation for the treatment of fractures was the traction method, which, properly applied, would prove efficient in the majority of diaphyseal fractures. It required skill and patience, and, if the surgeon could not employ it properly, it was unlikely that the leeway would be made up by skill in operation.

Examples of fractures of the shaft which required operation were certain fractures of the middle of the humerus and of the femur. If, after a fair trial by non-operative methods, co-aptation of the fragments could not be secured, there was certainly an interposition of muscle, a fact which rendered operation imperative. The intimate association of the triceps and quadriceps muscles in the two examples cited made this complication likely. Fracture of the shaft of the radius came into the same category.

If operation was to be performed in these cases what should be its nature? In his (the speaker's) own cases he had usually carried out simply an open reduction, using no internal fixation as a rule, or sometimes tying the two fragments with kangaroo tendon. He depended upon his assistant holding the limb until a suitable retentive apparatus had been applied. He avoided non-absorbable internal fixation as far as possible. If plates are used their removal might not be necessary later in the case of the femur, but in the case of the tibia they should be removed. In certain of the spiral fractures of the tibia Parham's bands acted very well. They, too, should be removed subsequently. Non-absorbable materials, such as plates and bands, did delay union in certain cases, and more than one case of delayed union and re-fracture had occurred in his (Mr. Cochrane's) experience from the atrophy endangered by their presence.

Protective splinting and walking caliper splints should therefore be employed for a longer period than usual. It was common for the fracture to bend at the site of union, or even to re-fracture if this was neglected, and it was a point worth emphasizing.

The most interesting personal series of cases he had had was that of seventy-four fractures of both bones of the leg. By far the greater number had been compound, owing to rupture of the skin by protrusion of bone. Such cases were only potentially infected and were not in the same category as war-wounds, in regard to which careful débridement was the essential factor in treatment. In the class of case in the series referred to, the use of antiseptics and débridement was not so important and the cases required a good deal of "letting alone" in this regard. In forty-four of the series, the wound had been enlarged and an open reduction performed, followed by fixation in plaster. None of the cases had become

infected and there had been no poor functional or anatomical results in the whole series. In only one of the cases had a plate been applied as a temporary means of fixation.

Mr. PAUL BERNARD ROTH said that during the last year at the Miller Hospital his colleague and himself had sent 2,400 cases for X-ray examination, and since the treatment of fractures had been in their hands they had been gratified by a large decrease in the proportion of bad results. He had arrived at the conclusion that operation should always be performed if there were displaced fragments which could not be reduced in any other way, even if this meant open operation. He did not always wish to operate, nor did he always wish to plate a case.

Operation should be performed as soon as possible after the accident. When a student, he had been taught that in fractured patella one must not operate until the whole of the swelling had subsided. He now thought that the proper time to operate on a fractured patella was the day following the fracture. If one waited, swelling would occur, but it would not occur if operation was carried out soon.

He did not think the presence of blebs constituted a contra-indication for operation. He now operated with perfect confidence, not in a state of apprehension of sepsis, etc.

One type of fracture for which operation should always be performed was fracture of the spine of the tibia. He operated within a week of the fracture, and replaced the fragment [slide shown]. If the fragment shown had been left there, the child would probably have been crippled for life. In this case the operation had been performed within a week of the accident, and the portion of tibia had been replaced.

Another type requiring operation was fracture of the lower end of the fibula where there was dislocation of the foot backwards owing to the triangular fragment of the tibia being displaced upwards. He had found no way of getting and keeping it reduced except by placing a screw through the fragment fixing it to the tibia. He operated on a foot by opening the ankle-joint following a method which Sir Frederick Eve had taught him. The incision was made on the outer side, the peroneal tendons lifted out, and the foot turned round to the tibia. The screw prevented dislocation from recurring.

Another kind of case in which operation was required was fracture of the lower end of the humerus. Attempts at reduction were made, usually without success. If the first attempt, made under an anæsthetic, did not succeed, no further time should be wasted on it. Exposure should be made from the outer side, the lower end of the upper fragment brought out, and cleared. It should be jammed into the cup-shaped lower fragment.

Dr. J. B. MENNELL said that some speakers appeared to indicate that all fractures of the lower end of the humerus involving the joint, and most, if not all, fractures of the head of the radius, should be operated upon. With regard to the latter fractures he had been dissatisfied with his own results because he had not secured complete extension rapidly, but on one occasion he had collected eight cases of fracture of the head of the radius, treated by mobilization and massage from the outset, and had shown them to Sir Robert Jones for criticism. The verdict was, that except in one case, the results were no less good than if operation had been performed.

Lucas-Championnière had never taught that one should not immobilize a fracture, or that splints should not be used. He had merely taught that splintage and fixation should be practised only to an irreducible minimum.

Finally, with regard to the treatment of fractures round the elbow-joint by early mobilization and massage, this should not be condemned because of its misuse. A point often overlooked was that if the joint surface had been open, there must be synovial fluid between the fragments. This invariably prevented the formation of any excess of callus as the result of early mobilization, unless the treatment were abused. Hence, when the joint-surface was open, free mobilization could be practised from the outset and it was often possible by this means to mould the joint-surfaces into perfect apposition. Operation on these cases, therefore, was frequently uncalled for, since a result quite as good could be secured without operation, and more quickly.

To treat a corresponding fracture in which the joint-surfaces had not been open, and in which there was no synovial fluid between the fragments would, of course, lead to disaster. Were this simple rule more generally recognized, an extended use of early mobilization treatment in cases of fractures of the elbow would be more greatly appreciated.

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Dr. SCUDDER (in reply) said that fractures had been treated for hundreds of years as they were in many instances being treated to-day, i.e., without reference to fundamental principles, and with varying results. The profession had now arrived at a point at which it had the experience of these hundreds of years *plus* the more vital and personal experience of many individuals in our own times. Consequently surgeons were standing, so to speak, on solid ground, saying that fractures were better treated now than they used to be, because, as a rule, knowledge was now based on acknowledged fundamentals. Surgeons might go on discussing their individual experiences, but unless they could co-operate and meet together for discussion no real progress would ensue. Surgeons were not doing all they might do in this respect, but a beginning had been made, as this meeting had shown. The work of Mr. Hey Groves, for instance, had carried the question definitely forward; his experimental work had demonstrated procedures which could now be carried out in human beings. In order to appreciate properly the merits of particular lines of treatment, more should be known of their functional end-results; at present surgeons were choosing methods based chiefly on impressions. He wondered how many of them followed up for a considerable time the cases in which they had operated, in order to learn what were the functional consequences. To know exact results in a few cases was infinitely more important than to derive impressions from a large series. The latter method was not scientific; efforts were made to compare two cases of fracture, but in reality they were not truly comparable. He had gone carefully over the material which the American College of Surgeons had collected recently, relating to cases of fracture of the shaft of the femur not involving the joint, and as a result an effort would be made to get the standardized hospitals of the United States and Canada to keep their records better and more exactly. Then a visitor to a hospital studying the question could have before him all the important facts resulting from treatment there.

Section of Anæsthetics.

President—Dr. Cecil Hughes, O.B.E.

[*May* 4, 1928.]

DISCUSSION ON LATE ETHER CONVULSIONS.

Dr. Charles F. Hadfield.

In the summer of 1926 there were several cases reported by the Resident Anæsthetist at St. Bartholomew's Hospital, of convulsive attacks during ether anæsthesia. Some of the patients had died either on the table or subsequently in the ward. The story was so contrary to my own experience, that I thought these must have been cases of epilepsy or some other condition unrelated to anæsthesia. Later, Mr. Wade himself had a case which impressed him so strongly that he immediately ordered that no more of the brand and batch of ether used in it should be issued.

The phenomenon in question is quite distinct from the familiar ether tremor or clonus. The latter develops early in anæsthesia and is dependent on or exaggerated by position. It may be troublesome but usually passes off early, and, so far as I am aware, is never attended by serious consequences.

The ether convulsions which develop later in anæsthesia are quite different. They usually begin with slight twitchings round the eyes or mouth, or both. These develop into convulsive epileptiform movements involving the whole body and sometimes violent enough to shake the operating table. The action of the cardiac and respiratory centres does not seem to be affected primarily, but soon becomes so from the violence of the movements, and may cease. If the anæsthetic is discontinued the movements may diminish and recovery follow. Unfortunately this is not always so, and death on the table has resulted. Alternatively the convulsions may continue, after the patient has been returned to bed, with a fatal result. Sometimes, again, they may return a few hours later and then cause death. Altogether the condition is very serious; I am so unfortunate, or rather so fortunate, as never to have seen a case.

Returning to Mr. Wade's case, immediate steps were taken to investigate the matter, and suspicion was first directed towards the ether, of the same make as that used in the previous cases. The makers at once undertook to assist the investigation in every way. Ether of the same batch was analysed in their laboratories and by independent analysts. At the same time some of the same ether was sent by us to Dr. King, of the Medical Research Council, whose extensive knowledge of the chemistry of ether is well known. All the reports stated that the ether was unusually good anæsthetic ether and free from any known impurity.

I collected what further information I could and reported fully to the Joint Anæsthetics Committee of this Section and the Medical Research Council. The committee determined to investigate the matter. It was, however, by no means obvious where to begin. The ether analysis led nowhere unless we assumed that the symptoms were due to some unrecognized impurity. This seemed improbable, not only on chemical grounds but also because although these cases all occurred with one batch of ether, the same ether had been used with hundreds of other patients without bad results. The makers sent me a consignment of this ether, and I used it for some time with no unusual symptoms and found it an excellent anæsthetic ether.

About this time the late Dr. S. R. Wilson sent many of us a questionnaire on this very subject. As there seemed to have been a good many cases in the Manchester district, he was in a better position to investigate than we were in London. Accordingly I suggested co-operation and sent him full details. His results were published in the *Lancet* the following May. With some of his conclusions we shall probably agree; if I criticize others, I do so with full appreciation of the care he devoted to this, as he did to all his other researches. He regarded these convulsions as due to some poison, and as the effects on the respiratory and circulatory centres seemed to be secondary, he concluded that the poison acted primarily on the nervous system. All the cases he records were in young people, and he pointed out that all the fatal cases—and most non-fatal ones—related to patients already toxæmic. He examined the ether used, and concluded that acetaldehyde and peroxide caused the convulsions.

I think this last and most important conclusion will hardly withstand careful examination. Although I am ready to admit that the convulsions may be precipitated or exaggerated by paraldehyde and such substances in the ether, it seems impossible that these impurities are the prime cause, though Dr. Wilson had excellent reason to suspect it. The ether used at his hospital seems to have been of poor quality. He tells us that as taken from stock, it contained "conspicuous amounts of acetaldehyde," and gave "definite tests as regards peroxide." He also found that "the amber glass bottles were spectroscopically efficient, but the glass of the storage cupboards, though apparently black, transmitted the full spectrum, especially the actinic rays." If the amber bottles were efficient the significance of the second remark is not obvious. The chemical examination shows that the ether was not up to the usual standard of anæsthetic ether. My committee has always found that the methylated ethers supplied for anæsthesia by the well-known makers are shown by the most stringent tests to be practically free from these impurities. Another point is worthy of mention, especially as some of Dr. Wilson's cases were collected from hospitals other than his own. From the London representative of a large maker of anæsthetic ether I recently learnt, to my surprise, that, contrary to advice, institutions of all sorts, more especially in the North of England, had purchased large quantities of relatively impure ether "not for anæsthetic purposes" for use as anæsthetic ether. Now, if institutions use, for anæsthetic purposes, ether which is definitely neither made nor recommended for such use, they must expect trouble. It is the duty of the anæsthetists at such institutions to protest. Although I do not admit that these convulsions are primarily due to impurities in the ether, it is likely that they are assisted by them. This use of inferior ether may help to account for the greater incidence of these convulsions in the North of England.

Again, if aldehydes and peroxides are the determining factors for these symptoms, why are the symptoms so relatively rare? Although good anæsthetic ether is practically free from these bodies when first opened, they soon appear, and then increase rapidly in quantity in the conditions to which it is frequently subjected. Some time ago Dr. King examined the ether remaining in my Boyle's machine after it had been used for several cases. Although it had been, at first, a good ether and presumably pure, Dr. King found it loaded with peroxide. One reason for the presence of large amounts of peroxides is their low volatility; as very little peroxide passes over to the patient it accumulates in the bottle as ether is added. The peroxides being little volatile are unlikely to cause convulsions. This would not apply to the aldehydes, which are rather more volatile than ether. These facts as to the formation of peroxides and aldehydes were confirmed and elaborated by Mr. Boyle, and published in the *British Medical Journal* in September, 1927.

The behaviour of ether under various conditions is often surprising. Some time ago, with Dr. King, I made some simple experiments. Taking two anæsthetic ethers of well-known makes, I passed nitrous oxide and oxygen through them for half an hour in a Boyle's machine under conditions comparable with those in an operating theatre, but varied as follows. In the first case nothing was done to prevent the natural cooling of the ether. In the second, the ether bottle was surrounded by a hot-water jacket. In the third, I placed a strong electric light with a reflector as close as possible to the ether, more than reproducing the strong light of an operating theatre, which has frequently been blamed for the formation of peroxides and other impurities. Each of the two ethers was subjected to these three treatments, and the resulting products, together with specimens of the ethers direct from the original bottles, were chemically examined by Dr. King. The results obtained and their consistency proved the accuracy of Dr. King's tests and his methods. They are as follows :—

	Ether X		Ether Z	
	Aldehyde	Peroxide	Aldehyde	Peroxide
Untreated	0	0	0	0
Ordinary light and temperature ...	3·3	5·5	0	0
Ordinary light, warmed	7·8	9·0	0	0
Electric light, unwarmed ...	3·2	4·5	0	0

These figures show conclusively that two ethers, as far as can be determined by the most delicate tests equally pure, react differently when subjected to a stream of gas and oxygen. The one remained pure in spite of the various treatments, while the other rapidly formed aldehyde and peroxide. Other important facts also emerge. It is obvious that the warming of the ether accelerates the formation of impurities, while exposure to strong ordinary electric light has no appreciable effect. Dr. King has, I believe, repeated the experiment, using a mercury vapour lamp, and found that it markedly encourages the formation of peroxides, etc. Another point of interest is the correspondence between the figures for aldehyde and those for peroxide.

These small experiments have no very direct bearing on the subject under discussion, but help us to appreciate the complexity of any problem connected with ether. They also confirm the rather surprising results reported to us some time ago by Professor Storm van Leuwen, of Leyden. He devised a means of preparing pure ether by the "ether of crystallization" method. He claimed that the ether so prepared was not only purer than that produced by any other method, but also that it would remain pure, although subjected to all sorts of ill-treatment, such as exposure to sunlight for long periods, even in uncorked bottles.

We see that much ether used to-day is likely to contain peroxide and aldehyde, yet the incidence of convulsions is far smaller than if they were directly due to these impurities. The impurities are probably more frequently present when ether is administered by methods in which gas and oxygen are bubbled through it. Yet cases have been noted with practically every ordinary method of administration. With one exception, to which I shall refer later, the convulsions do not seem to occur more frequently with any one method.

As far as I am aware, no case was recorded until two or three years ago. This does not suggest impurities in the ether. Since its first introduction as an anæsthetic, eighty years ago, ether has never been subjected to such careful chemical examination as during the last few years. Yet we are suddenly faced by these curious convulsions and asked to believe that they are due not to some hitherto unknown and unsuspected impurity but to such well-known by-products as peroxide and aldehyde.

An explanation was given for the sudden occurrence of these convulsions some two years ago. At the end of the War, the Government sold large stocks of excellent

ethyl ether to manufacturing chemists, who used them instead of making their own. Rather more than two years ago the Government stocks were exhausted, and the manufacturers resumed production of ether from methylated spirit. Almost immediately cases of ether convulsions began to be reported. I have tried to get independent confirmation of this story, but the only distinct answer I have obtained to my very guarded questions was a decided denial.

In the *British Medical Journal*, the same week that Dr. Wilson's communication appeared in the *Lancet*, another well-known Manchester anæsthetist—Dr. Pinson—published a paper on the same subject. He had collected the relatively huge number of fifteen cases, five of which proved fatal. Eleven cases had occurred in his own practice. All the patients were fairly young and the subjects of some more or less acute infection. Dr. Pinson is convinced that the convulsions are due to an excess of carbon dioxide in patients rendered particularly sensitive to it by ether and also possibly by the coexisting toxæmia. Any expression of opinion by such an authority as Dr. Pinson must be carefully considered, especially when he has had such a large and probably unequalled experience of the cases. Yet many other observers have noted that these symptoms are not ushered in by cyanosis—which, in fact, is not a feature until the convulsions begin to interfere with respiration. Again, Mr. Langton Hewer tells me that he has successfully treated the condition by free ventilation with oxygen combined with carbon dioxide. The disturbance is possibly due not so much to an excess of carbon dioxide as to a special sensitivity of the nerve cells to small quantities. Such a theory is at least as difficult to prove as to disprove and in the present state of our knowledge does not help us much Again, although the majority of recorded cases have occurred in septic patients, this is not invariably so. On the first appearance of any symptom Dr. Pinson advises immediate withdrawal of ether and the removal of masks and coverings from the face to assist the elimination of the carbon dioxide. After recovery, if a continuance of the anæsthetic is necessary, he recommends chloroform by the most open method. Here he differs from several other observers who have found that a change to chloroform forms the most certain means of precipitating a fatal issue.

Dr. Hornabrook is confident that the convulsions are due to excessive doses of atropine and states that for any patient over 12 years of age the dose should never exceed $\frac{1}{150}$ gr. atropine and $\frac{1}{8}$ gr. morphine. Harold Sington, however, shows that at Great Ormond Street Hospital more than 7,000 children are operated upon annually after doses of atropine greater than those mentioned above, and no case of convulsions has been observed.

A significant point is that these cases of late ether convulsions are almost confined to the warmer months of the year. It has certainly been so at St. Bartholomew's, where the original "epidemic" occurred in August and ceased as the cooler months came, although we had a few more cases the following summer. I regard heat, in some form or other, as the one definite, well-established factor. Dr. Wilson was satisfied that the steps he took to eliminate impurities from the ether produced the required result because the cases ceased. But by the time he had completed his investigations and carried out his reforms summer was already past.

In October, 1927, Dr. H. P. Fairlie suggested that the use of the ether bomb—as devised by Dr. Pinson—which delivers to the patient ether vapour at a relatively high temperature, might be a contributory cause. Dr. Pinson pointed out that some of his collected cases had not been anæsthetized by the bomb and that many others had been recorded when other methods of administration had been used. Still when we read that Dr. Pinson has had eleven cases in his own practice *all* of which were anæsthetized with hot ether vapour from the bomb, we must feel that the bomb may

be at least contributory. With this we can associate the increased incidence of these cases in warmer weather, and, possibly also, the fact that warmed ether is less stable than cold. Thus the one conclusion that emerges from these various considerations is that heat takes an active part in the causation of the symptoms.

Such a conclusion, unfortunately, does not help us very much. Every year many hundreds of thousands of patients are anæsthetized, many of whom are septic, many of whom are young or middle-aged and many of whom have their operations in warm weather or in overheated operating theatres. A smaller—but still very large—number of patients are anæsthetized with warm ether vapour from a bomb, a Shipway's apparatus or some similar machine. Yet the proportion of all these patients who suffer from late ether convulsions is so small that it probably could not usefully be represented as a percentage.

One other suggested cause of these convulsions was, I believe, first put forward by Dr. Mennell. He thought that the symptoms were those of a serious overdosage with ether. I do not think this is likely to contain the whole truth. There seems abundant evidence that many of the subjects of these convulsions were not at any time unreasonably deeply anæsthetized.

Lastly, I come to what I consider the most probable determining factor in producing these convulsions, vague and indefinite though it be. It is contained in that blessed word "idiosyncrasy." Possibly "diathesis" would be a better, though equally meaningless, word. I think there is evidence that there exist certain people possessed of some sort of idiosyncrasy or tendency which causes them to develop these symptoms when under ether. Probably in all cases it is necessary for one or more of several other conditions to be present at the same time. We had a case at St. Bartholomew's in which the patient was being operated upon for some thyroid condition. Convulsions began, the operation was stopped and the patient recovered. Some time later she was again anæsthetized with ether and again convulsions developed. This time the anæsthetic had to be continued and the anæsthetist, not knowing what else to do, changed the ether for chloroform, with a fatal result.

Some disordered thyroid secretion may conceivably have taken the place of the toxæmia so frequently found. Among Dr. Wilson's cases is one which is in some ways the converse of the last, but still may be taken as supporting the same conclusion. The patient had a compound fracture of the leg which was set under ether without untoward result. Gas gangrene developed, and three days later amputation was necessary. Ether was again given and this time convulsions and death supervened. This seems as if there had been the necessary idiosyncrasy but, at the first operation, without the accessory factor which the gas gangrene supplied at the second.

However, even if we provisionally accept this idiosyncrasy hypothesis, we are still faced by the more difficult problem of why these symptoms have only been seen—or at any rate recorded—within the last two or three years. Supposing that the manufacturers had ceased making ether for several years until the Government supply was exhausted: are we to believe that on resuming its manufacture, they had almost all become inefficient and made ether so bad that it caused serious symptoms such as had never been produced by even the worst ether made during the previous seventy years? This seems so unlikely that we can rule it out, quite apart from the fact that the ether associated with the symptoms has frequently been found to be chemically pure.

The only other possible suggestion is that these symptoms have always occurred sporadically but have only recently been described and recorded. This I cannot believe.

In conclusion I make five suggestions:—

38 Hadfield—Shipway—Daly: *Late Ether Convulsions*

(1) Some persons possess an unexplained tendency to develop convulsions under ether anæsthesia.

(2) One or more accessory factors may be necessary for the development of these convulsions in most if not all such patients.

(3) Among such factors may be found—possibly in order of decreasing importance: (*a*) heat; (*b*) sepsis or other toxæmia; (*c*) impurities in the ether; (*d*) youth.

(4) We know of no treatment except the withdrawal of the ether. The administration of oxygen, possibly combined with carbon dioxide, may assist. Chloroform should be avoided.

(5) We cannot at present explain the absence of any record of such cases previous to about 1926.

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Dr. F. E. SHIPWAY

said that some years ago Finnemore drew attention to the necessity of testing ethers in a paper read before this Section: he stated that the manufacturers were aware of the importance of this subject. He (Dr. Shipway) thought that out of Dr. Hadfield's valuable paper arose one outstanding fact, namely, that these convulsions had never been described until quite recently. Dr. Hadfield's table proved that ethers varied in quality and that a good ether could be thoroughly misused and maltreated without impurities being formed. It was known that once the formation of impurities began, the process went on rapidly: this process would of course be hastened by heat, and the method of giving ether by the Pinson bomb seemed, in this respect, faulty. He (the speaker) did not think this criticism applied to his own apparatus in which the ether was vapourized before being warmed. This method had been used in the War in thousands of cases of shocked, anæmic and toxic soldiers, without a single case of convulsions having been reported.

Dr. ASHLEY DALY.

Until 1926 when the late Dr. S. R. Wilson published his paper on this subject. I had neither heard of the condition nor had a case in my own practice, but three cases have occurred at the London Hospital, of which I treated the first.

This case seems to bear out all Dr. Wilson's conclusions:—

Patient, a thin, spare woman, aged 30. Operation, partial gastrectomy for ulcer high up on lesser curve. Anæsthetic: A.C.E., followed by ether, first on gauze mask, later by Shipway's inhaler, the ether being vapourized with oxygen. Convulsions severe and general came on after forty minutes. I saw the case after convulsions had started and at once gave carbon dioxide 20 per cent. in oxygen in gas bag; this caused the convulsions to cease after about three minutes; the patient made a good recovery though there was severe vomiting for two days, and during this time the urine contained acetone and diacetic acid.

Examination of the ether in the Shipway inhaler, which had also been used for a previous case lasting thirty-five minutes, showed the presence of acetaldehyde and peroxides; examination of ether from which the inhaler was filled showed no acetaldehyde or peroxides.

In both the other cases there was appendicitis, in one with abscess, in the other with slight inflammation; in neither case was a Shipway inhaler used, the anæsthetic in both cases being open ether. In both cases the patient recovered.

I do not agree that warming ether is a contributory cause; one of these cases occurred in July, the other two in March. I think the cause may be found in some impurity in the ether, possibly also in very deep anæsthesia.

Dr. L. Kirkby Thomas

described five cases which he had seen between the years 1913 and 1925, and expressed his firm conviction that, apart from the question of impurities in the ether used, the symptoms observed were the result of over-etherization, and could be avoided by careful and judicious administration.

Dr. Z. Mennell

said he had had experience of three such cases in his own practice, two more he had been called in to see at St. Thomas's Hospital, and he knew of three others. In all these cases the patients had been deeply anæsthetized and had been flooded with oxygen. The convulsions had occurred late and during the use of three different brands of ether.

His first experience, six years ago, had occurred in the winter during a difficult gall-bladder case in a middle-aged woman, who had recovered, but had died three days later from acidosis. The convulsions had begun in the usual way, with twitching at the corners of the mouth, when the patient was deeply anæsthetized and flooded with oxygen.

For the last two years he had not seen a case, though sometimes the same necessary extreme depth of anæsthesia had been procured, but without oxygen. He considered that extreme depth of anæsthesia with over-oxygenation was a cause of these conditions. They all occurred when blowing a continuous stream of oxygen through ether; not a single case had occurred when air had been substituted for oxygen. Surgeons nowadays demanded a much deeper anæsthesia than formerly, and they obtained what would have been regarded as wonderful relaxation in consequence.

He was not prepared to say whether the convulsions were due to—

(1) Over-oxygenation. (2) The formation of impurities in the ether from the use of oxygen. (3) The ability of the nerve cells in the medulla to absorb more ether in the presence of an excess of oxygen; but that these convulsions were in some way associated with over-oxygenation seemed to him to be apparent.

Mr. H. E. G. Boyle

said he had not had any of these cases in his own practice, but had seen the conclusion of one at St. Bartholomew's Hospital, and his impression from this case, and the reports of other cases, was that all the patients appeared to have been deeply under ether, and he agreed that this might have been the cause of the convulsions.

There was one point on which he would like some information, If ether was given to a dog to which morphia had been previously administered, there was less liver change than if the morphia had not been given. Had the patients in any of these cases had morphia administered to them beforehand?

He had had some exhaustive experiments carried out on peroxides and aldehydes. The conclusion amounted to this: If peroxides and aldehydes were present in the ether, then bubbling nitrous oxide and oxygen through it produced these peroxides and aldehydes in proportionally increasing quantity, but if they were not present to begin with, then very few peroxides and aldehydes were formed. Whether this impurity was the cause of these convulsions he was not prepared to say, but the resulting mixture had a most pungent and irritating smell.

40 Featherstone—McDonald: *Late Ether Convulsions*

Dr. Henry Featherstone

said that his colleague, Dr. McCardie, regretted his inability to be present. Dr. McCardie wished it to be known that in thirty-two years of practice in anæsthetic work he had not encountered a case of convulsions under ether anæsthesia. Dr. Hassall and the speaker, in their work at the Birmingham General Hospital and elsewhere, had not seen a true case of ether convulsions. He (Dr. Featherstone) was inclined to attribute such convulsions to impurities in the ether, which might be due to the method of administration, such as the ether-bomb method, or, as in a recent case, the ether might be impure. The case in question occurred at the Children's Hospital, Birmingham, during the previous week. Analysis proved that the ether contained peroxides, aldehydes, and other impurities. The patient was a boy aged 8, suffering from pneumococcal peritonitis. There was no history of epilepsy. Ether was administered by the open method and there was no convulsive symptom until the abdomen was opened. The convulsions were of the clonic type, were not controlled by deepening or by lightening anæsthesia, or by the administration of oxygen, and occurred in bouts at short intervals. Bromide was used with some success, as the convulsions ceased about two hours after returning to bed. The child vomited freely, but owing to his disease it was not possible to estimate the after-effects. At the time of speaking he was still alive.

It was interesting to note that the child was young and toxic, because Dr. Hadfield believed youth and toxæmia to be possible ætiological factors.

Dr. Niel McDonald.

Two cases of ether convulsions occurred in my practice during summer of 1927:—

(I) At the Hospital for Sick Children, Great Ormond Street, London, in a girl aged 2 years, operated on for cicatricial contracture of fingers following burns. Anæsthesia induced by ether on mask and maintained by bubbling oxygen through warmed ether in a Boyle's bottle. Slight, but typical "ether convulsions" began after about one hour's anæsthesia and towards the close of the operation. Ether was then stopped and oxygen alone was given and the convulsions ceased ten to fifteen minutes afterwards and recovery was uninterrupted. I have since anæsthetized the same child twice with no untoward occurrences.

(II) At a nursing-home, in a girl aged 6 years operated on for subacute appendicitis. Induction by ethyl-chloride; anæsthesia maintained by ether on mask covered by towel, oxygen being delivered under towel direct from cylinder. Convulsions, again slight but typical, came on about half an hour from start of operation, and during closure of abdominal wall. Ether was withdrawn and oxygen alone given, convulsions ceasing after about ten minutes; recovery uninterrupted.

These patients were anæsthetized by ether from different makers, but analysis in each case showed traces of peroxide in the samples used.

It has been my custom for a number of years to employ very deep ether-anæsthesia combined with continuous administration of oxygen in a large number of cases, but I have only seen the two cases of ether convulsions mentioned above. I therefore differ from those who think that either deep anæsthesia or over-oxygenation is the cause of the convulsions.

Section of Dermatology.

President—Dr. J. M. H. MacLeod.

[*May* 17, 1928.]

CASES.

Morphœa with Root Distribution.

By H. W. Barber, M.B., F.R.C.P.

A. D., aged 15. For three years the patient has had this condition. The area of skin involved extends round the left side of the chest following the line of the lower intercostal spaces, corresponding to the distribution of an eruption of herpes zoster in this situation. Although the affected skin cannot be said to be sclerodermatous, the bluish-brown discoloration resembles that which follows patches of morphœa, and it seems probable that at one time there was true scleroderma.

Mr. Blake kindly carried out some investigations for me on the sympathetic nerve supply of the area as follows :—

General Sympathetic.—Pupils and heart react normally.

Local Reactions.—The red tache can be obtained in rather modified form over the area of scleroderma. The white tache cannot be obtained clearly over the pigmented area, but is apparent on adjacent skin.

Reaction to Pilocarpine.— $\frac{1}{4}$ gr. injected into the deltoid did not produce definite sweating on the chest. $\frac{1}{8}$ gr. produced well-marked sweating over the chest in about half an hour. Sweating was noticeably decreased over the pigmented area.

Psycho-galvanic Reaction.—Leading off from both hands a fairly well-marked fall of resistance was obtained on a sensory stimulation.

Dry Heat.—Sweating was not well marked, though radiant heat was applied to the chest for nearly forty minutes, until it was distinctly uncomfortable.

Summary.—The sympathetic system as a whole seems fairly normal. The sympathetic fibres to the chest seem to be rather inefficient. The sympathetic supply to the pigmented area is deficient, and the surface capillaries in that area are sluggish in their reaction.

Similar investigations were carried out for me by Dr. Millis in another case, a girl with scleroderma, involving the deltoid, suprascapular region and the inner side of the arm, associated with some muscular atrophy and paresis. In this case the causal factor was a lesion of the brachial plexus, and there was very definite evidence of involvement of the sympathetic fibres supplying the affected area of skin.

Discussion.—Dr. Arthur Whitfield said that long ago Sir Lenthal Cheatle had put forward a good many cases which Sir Henry Head had accepted as sclerodermatous areas on "maximum points." He (the speaker) was not sure whether the present case was one of sclerodermia.

Dr. Graham Little said he considered that the atrophic patches were on the points of maximum distribution of a herpetic eruption, and that led him to think this case was herpetic. The eruption had the distribution of the metameric segments of Head.

Case of Streptothrix Infection (previously shown).

By H. W. Barber, M.B., F.R.C.P.

K. M., aged 32, a Japanese man, was previously shown as a case for diagnosis at the Meeting, December 15, 1927.[1]

The President had then suggested that the condition was due to a streptothrix infection, a view which has since been confirmed. The condition at that time is seen in the accompanying photograph. The affected foot became secondarily infected with a hæmolytic streptococcus and the patient was very ill, with a high temperature lasting several weeks. Mr. Davies Colley operated and made several incisions. Some of the pus was of the granular type, such as is seen in actinomycosis, and in stained preparations of this Dr. Knott was able to demonstrate a streptothrix, but all attempts to culture the organism have failed.

The patient's condition became progressively worse and he was advised to have the foot amputated. This he refused to sanction, and it was thought he would die. French tincture of iodine was given in large doses and gradually the temperature fell, his general condition improved and the foot began to heal. Not only that, but the other granulomatous lesions on the legs and shoulder have cleared up, leaving scarring.

I attribute the remarkable result to the iodine: he had previously been on potassium iodide, but no substantial improvement was manifest until the French tincture of iodine was given.

(I) Leuconychia with Dysidrosis; (II) Leuconychia.

By S. E. Dore, M.D.

These cases are well-marked examples of total or partial leuconychia, lasting, in each case, five years, and both the patients are butchers. Another case of the kind was shown recently by Dr. Graham Little, and the patient in that case was also a butcher. In one of these cases the patient ceased work for three months and during that interval, it is said, the nails became normal, relapsing when he resumed work. It looks, therefore, as if this condition can be classed among the industrial diseases. Both these men say their hands are not unduly immersed in water, and that they do not sustain any particular injuries in their work. However, as Dr. Whitfield has pointed out to me, they are constantly handling moist meat. In both cases some of the toe-nails appear to be whiter than normal, but are not definitely affected. There are two sisters and a brother who are said to be unaffected. One of the patients also suffered from dysidrosis, but that has now cleared up.

Discussion.—Dr. J. M. H. MacLeod (President) said the main point was to decide whether this condition had developed because of the occupation, or whether it was familial leuconychia. He had known one or two cases of the latter condition.

Dr. W. J. O'Donovan said that Dr. Dore's two remarkable cases brought to mind a similar case of leuconychia that he (the speaker) had seen at the London Hospital last year. This patient was a butcher who had cut and pickled meat for many years. The leuconychia had lasted for some five months without other skin affection and the patient connected this

[1] *Brit. Journ Derm. and Syph.*, 1928, xl, 168.

with his occupation. Owing to a lack of similar cases he had not been able at the time to agree with the patient's suggestion, but these two cases of Dr. Dore's added much weight to the opinion that occupation was an important etiological factor.

Dr. W. KNOWSLEY SIBLEY said he had seen some half-a-dozen cases of leuconychia, and he had always considered it familial and congenital. In 1911 he had published, in the *British Journal of Dermatology*, a case of the kind, and he recalled the case of a young girl, apparently quite healthy, who had lichen planus, and she had had marked leuconychia all her life. In the cases he had seen the condition seemed to have been independent of occupation, and to have run in families. He had never known a recovery in these cases, but he considered that the condition was not so rare as was generally supposed, as it was often overlooked because patients affected with it did not present themselves for treatment of this condition, but for other diseases, when it might be accidentally observed.

Dr. ARTHUR WHITFIELD said that four cases in butchers had now been mentioned. If this was an occupation condition—an assumption supported by the statement that after three months' absence from work it disappeared—he thought it must be due to poison working from within, rather than from without. Preservatives were not supposed to be used for meat, and he did not ask the patients whether they used preservatives, but if they were used he did not see how a fundamental change could occur in the matrix without affecting the posterior nail folds, which were flat and adherent, not œdematous. This might be a toxic condition which affected only a few of the people in the meat trade.

Parakeratosis Variegata or Parapsoriasis Lichénoïde (Brocq).

By J. M. H. MACLEOD, M.D. (President).

THE patient is a tram driver, aged 42, and the eruption, which is chiefly on his trunk, arms and legs, and to a slight extent on the face and backs of the hands, appeared six years ago, attacking the whole of the affected skin about the same time. Since then it has persisted and is unaffected by treatment. It is not associated with itching, unless subjected to friction or heat, and the general health of the patient is unimpaired.

The case closely resembles one described by the exhibitor and the late Dr. Colcott Fox in 1901, and reported in the *Journal* of that year.

The eruption is of the mottled, retiform type of parapsoriasis. The essential lesions consist of irregular macules, or maculo-papules, about the size of a pin's head. These tend to coalesce and form a pinkish network, enclosing areas of normal skin, the meshes of the network averaging 2 mm. in diameter, and being polygonal in shape. The epidermis over the flat papules is either stretched (the stretchings giving it an atrophic appearance), or it forms a scale. The skin of the palms, though dry and thickened, is not affected, nor are the nails involved.

The ætiology of this condition is still unknown. It seems to occur chiefly in adult males. In one case recorded by the exhibitor it occurred in an aeroplane test pilot, who attributed it to flying at great height. In another, recently shown before the Section, it was associated with a vasomotor rhinitis, due to protein sensitization. Brocq regards it as allied to psoriasis, while the late Dr. Crocker suggested that it might be connected with exposure to high temperature, as he had seen two cases in furnace men and one in a fireman.

Dr. Colcott Fox, with his accustomed care in regard to nomenclature, insisted on the condition being called a "resistant maculo-papular erythrodermia." He had seen in Edinburgh a case which was described by Unna as "parakeratosis variegata," which he believed to be the same as the case we described.

Discussion.—Dr. GRAHAM LITTLE said the acute onset of the case interested him. He had had a patient, a young girl, in whom the condition had come on so suddenly that the local doctor had regarded it as an exanthem. When he (the speaker) had shown her before the Section the condition had persisted for six years, and the late Dr. Pringle had said he had seen the same condition several years before, and Dr. Jamieson had referred to it in his paper. In all the cases he (Dr. Graham Little) had seen, the conditions had remained totally unaffected by treatment.

Dr. F. PARKES WEBER asked whether blood-counts had been made in cases of parakeratosis variegata like the present one. Sequeira and Panton, in their paper on "Lymphoblastic Erythrodermia," included the case of an elderly man, said formerly to have had "parakeratosis variegata."

Dr. ARTHUR WHITFIELD said this was only the second case he had ever seen of the kind; the first was that seen by the President in conjunction with Dr. Colcott Fox. As soon as he saw the present case he recognized it as the same condition. He believed cases had been carelessly described as parakeratosis variegata which neither the President nor Dr. Colcott Fox would have accepted as such. The case mentioned by Dr. Parkes Weber was probably not very well defined. The eruption in the present case was most characteristic.

Dr. MACLEOD (in reply) said that the blood-count revealed no significant change in the first case, and in the present case it had not been taken. He knew the type of case referred to by Dr. Weber—lymphoblastic erythrodermia—and he had seen a similar skin condition in association with Hodgkin's disease, but the appearance of the skin in such cases was different from that in this case.

Erythematoid Benign Epithelioma.

By E. G. GRAHAM LITTLE, M.D.

THIS is an example of a rare condition, namely, a malignant carcinomatous change upon the site of an old-standing basal-celled epithelioma, of the flat, erythematoid type. The man has three lesions, all with the same characteristic appearance, the oldest, eight years in duration, being the one in which the malignant change is taking place. They are all on the back, which is one of the sites of election. The two other lesions have undergone an entirely different evolution: in one there is very little left, the other has entirely disappeared, with cicatricial atrophy. Before excising the patch in the present case I thought I would show it to the Section. I do not think it is more common to see malignant change in these flat basal-celled epitheliomata than in lupus erythematosus for example. In 1908 I saw a man with a flat basal-celled epithelioma on the back of his trunk, from which a section was obtained. The lesion gave him no trouble, and I heard no more of him for six years, when he told me that he had had no further trouble. In 1922 he came up again, this time with an undoubted squamous-celled carcinoma on the old flat basal-celled epithelioma. It was excised by Mr. Warren Low, and the sections of the growth made in 1908 and those made in 1922 are in the St. Mary's Hospital Museum, and show very strikingly the change which has taken place.

Discussion.—Dr. G. B. DOWLING said that three years ago he had had a case of flat basal-celled epithelioma of many years' duration, in an old man. On one portion of the growth was what appeared to be a large squamous-celled carcinoma, and there were enlarged glands in the axilla and other parts. The patient had died from carcinoma. Sections were cut, both of the new active growth and of the old basal-celled growth.

The malignant portion of the growth was not an ordinary squamous-celled carcinoma, but was composed mainly of cells decidedly smaller than those usually found in this type. They appeared to represent a transitional stage between the basal-celled type of growth and the squamous-celled type. The flat portion of the growth appeared also to be undergoing this change, although clinically no obvious distinction was apparent.

Dr. W. J. O'DONOVAN said that he had seen two similar cases of chronic superficial basal-celled epithelioma. In the first (published in the *British Journal of Dermatology*, with a coloured plate by Dr. Sequeira) the growth had been on the back. In the second, which was under his (the speaker's) care at present, the growth was on the lower abdomen.

With regard to the natural spontaneous cure of malignant disease of the skin, he had under his care at present a man whom he had first seen with a carcinomatous ulcer on his scrotum, which had completely disappeared, leaving a supple white scar, while an exuberant mass of breaking-down secondary growth had developed in the left groin.

Dr. J. A. DRAKE said he had seen a case similar to this in a man aged 80, who had a number of benign erythematoid lesions scattered over his body. Some were typical, but others were so small that one could scarcely recognize them. In the middle of his back the man had an apparently carcinomatous ulcer of the same appearance as that in Dr. Little's case. One or two of the original lesions had disappeared spontaneously, leaving a smooth scar.

Dr. H. MACCORMAC said that he had exhibited at a meeting of the Section a patient with a number of superficial benign basal-called epitheliomata of the type shown by Dr. Little, in one of which a part of the growth had become more active, forming a warty mass. He had also had similar cases in his practice (including that mentioned by Dr. Little as having been under the care of the late Dr. Pringle), and he did not think this modification of the growth was very uncommon. He asked Dr. Little why he applied the term "benign" to these superficial forms of rodent ulcer? If they were genuine examples of rodent ulcer—and this was generally accepted—then they were not "benign," but cancerous or malignant tumours.

Dr. WHITFIELD said he could not speak quite so confidently as Dr. Little, as to the benignity of these tumours. Of the numerous cases of lupus erythematosus which he had had under his own care none had developed malignancy, whereas of the something less than a dozen of the epithelioma cases one had developed a regular carcinomatous change. Nevertheless he was prepared to agree with Dr. Little that the majority of patients affected by these tumours passed through life without any serious trouble from them.

Dr. GRAHAM LITTLE (in reply) said that the question of malignancy was relative. Relatively, this was an astonishingly benign form of rodent ulcer; the late Dr. Pringle had had a case similar to this now shown, which had lasted fifty years, and the speaker had himself had a case of a flat spreading rodent which had lasted forty-five years. Some years ago he had shown an old lady who had been operated upon by Mr. Openshaw for Paget's disease of the breast. On the site of that operation a rodent ulcer of this type had developed: it began twelve years ago; it was very superficial and had spread until its area was 12 in. by 8 in. There had, however, been no malignant change, but merely a very slow extension, and for that reason he spoke of the process as "benign." In a number of cases the growths underwent spontaneous involution, leaving a small atrophic scar as the only trace of the pre-existing lesion—a further argument in justification of the distinction of this type of rodent as essentially benign.

Dr. MACCORMAC said that the discussion turned upon whether the lesions in question were or were not rodent ulcers, because a rodent ulcer was a malignant new growth. It appeared to him that the expression "benign erythematoid rodent ulcer" was contradictory. The slow development, characteristic of the lesions, although a striking feature, did not alter the fact of their malignancy.

Dr. WHITFIELD said he objected to the idea that a definite criterion of malignancy could be established by microscopical examination, "Malignant disease" was in reality a clinical term. If he asked a pathologist why he called a certain tumour malignant, the answer was

that, from a wide experience of microscopical appearances and the known clinical course, he (the pathologist) frequently knew that such a growth would cause serious illness or death.

If the pathologist affirmed that invasion of deeper tissues by epithelial growth was a criterion of malignancy, what was the position with regard to a developing mole or Brooke's adenoma cysticum? Both invaded the true skin and both might develop undoubted malignancy, but neither was classed as malignant. It was only when these tumours started into active growth and became progressively destructive that one classed them as malignant, and this was a part of the clinical course. Many "rodent ulcer" tumours remained as flat buttons, without either extension or destruction, for long periods, or for the whole of the subsequent life of the patient.

He (Dr. Whitfield), from long experience, might say whether a mole was malignant or not, and in most cases would be right, but he was willing to admit that if a mole, e.g., in the axilla, were subject to irritation, and the section showed inflammatory reaction in consequence, he would be unable in some cases to give an opinion.

Dr. MACCORMAC said he could not agree with Dr. Little that malignancy was a relative term. Dr. Whitfield would agree that rodent ulcer possessed recognizable and recognized clinical and pathological features which placed it among the category of malignant new growths. It therefore seemed illogical to label any condition definitely of this nature as "benign" if the term benign was employed in its accepted sense, whether one was a pathologist or a dermatologist. He failed to see the exact bearing on the subject under discussion of Dr. Whitfield's observations upon moles. Moles were nearly always simple, although on very rare occasions they might develop into the most malignant of all forms of malignant disease. They were, in fact, either simple or malignant at any particular moment, and the inability of the observer to interpret, in difficult cases, their simplicity or malignancy did not alter this fact.

Dr. GRAHAM LITTLE (in further reply) said it was no more necessary or probable that these flat rodents would become what was called malignant, than that other conditions, termed —perhaps unhappily—"pre-cancerous," would do so. He contended that it was justifiable to call "benign" a condition which persisted fifty years, as in Dr. Pringle's case, without any tendency to become clinically malignant. It was an appropriate term relatively to the clinical behaviour of the case. He was glad to have the support of Dr. Whitfield in the application of the term in the sense in which clinical dermatologists, as contrasted with morbid anatomists, used it.

Dr. J. M. H. MACLEOD (President) said that he had seen two cases in which the flat type of rodent became fungating and developed into squamous-celled epithelioma. In one case in which this had occurred, as the lesion was too large for excision, he had obtained fairly satisfactory results by curetting, followed by X-rays. As a result of this treatment, the condition had remained healed for two years. Any slight crusting that had since developed had been dealt with satisfactorily by freezing with carbon-dioxide.

Papulo-vesicular Eruption with Secondary Streptothrix Invasion.

By H. MACCORMAC, C.B.E., M.D.

THIS patient first came under my observation about a fortnight ago, having a discrete papulo-vesicular dermatosis distributed over the extensor surfaces of the forearms, and also present in a slight degree on the back of the neck and right eyebrow.

He is by occupation a horsekeeper, and was advised to attend hospital by a veterinary surgeon, as one of the horses under his charge had recently died of "purples" (infective purpura). As the lesions he exhibited were of a rather unusual type it was decided to have their contents examined bacteriologically. Dr. Whitby carried out the investigation, and from an apparently uncontaminated vesicle obtained a streptothrix in pure culture.

I believe that no similar case has previously been shown before the Section. In my opinion the organism stands in no direct causal relationship to the eruption, and is to be regarded rather as an accidental invasion by a non-pathogenic—or only very slightly pathogenic—organism, probably derived from the fodder in which the patient is constantly working. This view is supported by the fact that the eruption has rapidly improved under simple calamine lotion, although consideration should be taken of the manner in which the parasite was cultivated directly from the apparently uncontaminated vesicular contents.

Lupus Annularis.

By R. T. Brain, M.D. (for Dr. W. J. O'Donovan).

O. A., a single woman, aged 43, first attended the London Hospital in July, 1926. Three years previously a small pimple appeared on the right side of the nose and slowly enlarged to produce a ringed lesion which extended over the bridge. In spite of treatment at two other London hospitals the primary lesion continued to grow and several new ones appeared on the forehead (fig. 1). The patient states that these later lesions had ring-like characters from the beginning.

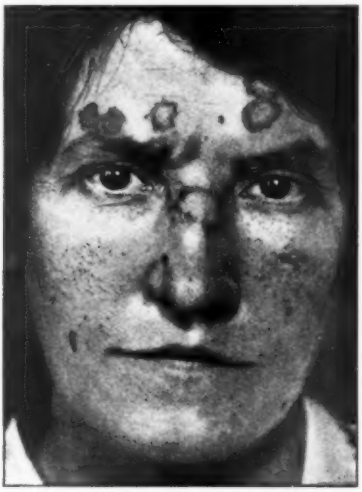

Fig. 1.

There was no familial or contact history of tuberculosis, and the patient, a domestic servant, enjoyed good health.

The photograph shows her appearance in July, 1926. There were seven annular lesions on the forehead and nose, with a few adjacent pinkish-brown, rather translucent nodules. The lesion on the nose showed a pale, smooth central scar surrounded by a raised reddish brown margin, which appeared to consist of contiguous,

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yellow, translucent nodules, 2 to 3 mm. in diameter, in an erythematous matrix. The other annular lesions were similar, although their centres were not so pale, because the scarring was slight, and the margins were less nodular.

The lesions most resembled lupus vulgaris, but the peculiar brownish tint and the remarkable annular characters, together with the relatively slight degree of scarring, made the diagnosis suspect. The Wassermann reaction was negative.

In October, 1926, a nodule was excised for histological examination. The sections showed a granulomatous inflammation of the skin extremely suggestive of tuberculosis. There were many well-defined areas of infiltration with epithelioid cells, and here and there a characteristic giant cell or a diffuse necrosis completed a very typical picture. One of these areas, drawn with the aid of a camera lucida, is shown (fig. 2).

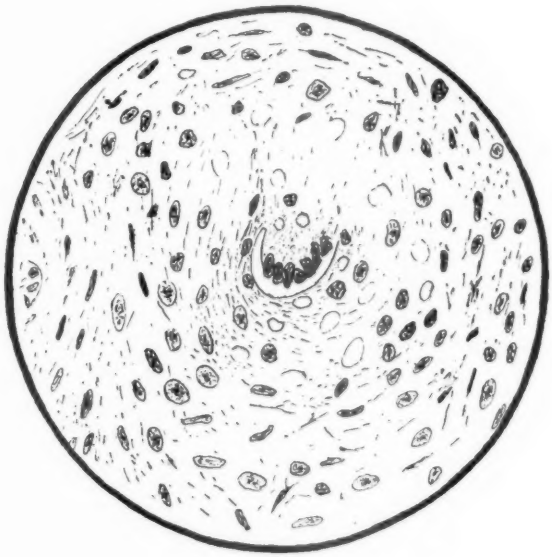

FIG. 2.

A number of sections have been examined for the presence of tubercle bacilli but without success.

There are very few references to lupus annularis in the literature. Stelwagon, 1921, briefly refers to the condition in his book, and quotes the case reported by Elliott in 1896, of a female, aged 47, with a two years' history. There were fourteen ring-shaped lesions on the face consisting of a white, shiny, smooth and entirely cicatricial central area surrounded and limited by a narrow band, one-sixteenth to one-eighth of an inch in breadth only, slightly elevated and unbroken—perfectly smooth, of a brownish colour and giving the impression of translucency. No separate nodules were present. The microscope diagnosis was lupus vulgaris and tubercle bacilli were found in the lesions. The above description is exactly that of

our own case, with the single exception that in many of the lesions the central scarring was much less marked.

Ransom and van Gieson, in 1895, reported a similar case, but the sections were not typical of lupus vulgaris although tuberculosis was produced in guinea-pigs by inoculation. The only other reference to lupus annularis found in the literature is by Sutton, in 1910. In his case there was a solitary lesion on the thigh. The sections were typical and tubercle bacilli were seen.

REFERENCES.

G. T. ELLIOTT. *Journ. Cutan. and Genito Urin. Dis.*, New York, 1896, xiv, 476. C. C. RANSOM and I. VAN GIESON, *ibid.*, 1895, xiii, 269. R. L. SUTTON, *Journ. Cutan. Dis. incl. Syph.*, 1910, xxviii, 391. H. W. STELWAGON, "Treatise on Diseases of the Skin," 1921, 758.

Discussion.—Dr. W. O'DONOVAN said that in his collection of photographs he had only one of a similar condition which had lasted for many years; the patient in that case had died recently from tubercular meningitis, and, post-mortem, large calcareous nodules had been found in the lungs. In the present case the infiltration and scarring were minimal, but when first seen the lesions were thicker, more raised, and broader. In the left eyebrow were discrete apple-jelly nodules. The microscopic appearance seemed to be that of lupus and he saw no reason for diagnosing the case as anything but a form of multiple lupus of very mild pathogenicity, in which the patient's powers of resistance were rapidly and very successfully heightened by general light baths.

Dr. H. W. BARBER said he thought this case was typical of a certain variety of sarcoid. He had recently shown a similar case before the Section. The lesions healed up in the middle, leaving central scarring, and spread with a lupoid edge. A section was submitted of this case, and most Members agreed that the appearances were those of sarcoid. He had had two other cases, one of which he had also shown. He asked Dr. O'Donovan, with regard to his fatal case, whether the lesions had disappeared before death. According to Schaumann, if phthisis developed in a patient with sarcoid, the skin lesions automatically disappeared. Had the von Pirquet test been done in the present case? Unless visceral tuberculosis was present, the reaction should be negative in sarcoid.

Dr. J. M. H. MACLEOD (President) said he regarded the case as a peculiar type of sarcoid.

Dr. O'DONOVAN (in reply) said that it was a tradition of the London Hospital Lupus Department, which he had himself confirmed, that if lupus disappeared with undue rapidity, the development of fatal visceral tubercle was to be expected. To him, therefore, and to his fellow-workers, the rapid disappearance of what appeared to be lupus was in no way a suggestion that the diagnosis of lupus should be altered to that of sarcoid.

The von Pirquet reaction had dropped out of use as a routine measure for the differential diagnosis of difficult adult cases. It had not proved helpful in the solution of clinical problems.

Case of Hyperkeratosis Palmaris et Plantaris associated with Ring-like Constriction of the Fingers.

By J. E. M. WIGLEY.

THIS girl, aged 10, has marked hyperkeratosis of palms, palmar surface of fingers of both hands and soles of both feet. Over the knuckles and interphalangeal articulations are similar hyperkeratotic lesions, each having a star-shaped appearance. There are two similar lesions over the right knee. These lesions are said to have been present since the age of three months.

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Round the middle phalanx of each little finger is a constricting fibrous band which has led to almost complete amputation of the left finger. Radiographic examination of these fingers shows atrophy of the phalanges, that of the left side having practically disappeared. These constricting bands appeared at the age of seven.

There is no history of any other members of the family, as far back as the grandparents, having any similar affection. The child is the only member of a family of four who has any physical defects, and apart from this she herself appears normal.

She came to Dr. MacLeod's Clinic to-day for the first time. It is hoped to investigate the condition fully and report on it later, as the combination of lesions is very unusual. (I am indebted to Dr. MacLeod for permission to show the case.)

Discussion.—Dr. J. M. H. MACLEOD (President) said the case reminded him of a condition known as " mal de Meleda " (from an island on the Dalmatian coast), in that the hyperkeratosis was not only on the palms and soles, but on the knuckles and elsewhere. The association of the peculiar " ainhum " condition in the present case was specially interesting, and suggested the possibility of ainhum being a congenital defect.

Dr. S. E. DORE said he had seen several instances of the condition mentioned by the President, one in both a father and a daughter. He had also seen a child about the age of this girl and one little finger was affected in the same manner. He only saw the case once, and had not been able to follow it up.

Section for the Study of Disease in Children.

President—Mr. GEORGE E. WAUGH, F.R.C.S.

[*May* 18, 1928.]

The Clinical Aspect of Congenital Mesenteric Malformation in Children.

PRESIDENT'S ADDRESS.

ABSTRACT.—(1) Congenital malformations of the mesentery are a definite morbid entity of a chronic type which may be recognized, before operation, by careful clinical investigation. (2) The symptom-complexes to which they give rise cannot be explained by reference to any of the well-known abdominal surgical diseases; still less by any purely functional disability which may be included under the term "indigestion." (3) The most important physical sign is the "emptiness" of the right iliac fossa, associated sometimes with an asymmetrical enlargement of the abdomen on the left side. These signs follow of necessity, inasmuch as the whole segment of the embryonic mid-gut is involved in a failure of rotation and fixation after reduction from the umbilical sac. (4) Radiological investigation should prove to be more helpful in confirming the clinical diagnosis when the special method of examination already described is used as a routine. (5) Operative treatment may cure the patient; alternatively it may reveal a pathological condition for which a rational course of treatment may be drawn up subsequently, when the precise details of the malformation have been discovered.

THE complicated evolutions which the fully developed mid-gut segment of the alimentary tract undergoes in its course of reduction from the primitive umbilical sac to the abdominal cavity proper, during about the eighth week of fœtal life, must proceed unchecked in any detail before the normal architecture of the alimentary tract can be accomplished. The different phases of this evolution are reduction, rotation, descent from a higher to a lower plane of the abdominal cavity, and, finally, fixation. At one end of the pathological scale is the condition of exomphalos in which no departure has been made from the primitive embryonic condition of the eighth week, and the child is born with the whole of the mid-gut loop present in a persistent umbilical sac. At the other end of the scale the whole process has been carried out successfully, with the exception of fixation, which is illustrated by the persistence of a primitive vascular mesentery to the whole of the ascending colon. In between these two extreme types, all forms of malformation dependent upon the failure of completion of one or other of the different phases of evolution have been described; these descriptions, however, have not been based upon clinical diagnosis made beforehand upon children suffering from vague abdominal disorders, but rather from deliberate embryological studies made in the post-mortem room, or from accidental findings during the course of abdominal operations undertaken for other

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conditions, or at operation for acute abdominal catastrophes in early infancy—such as volvulus—which have been surmised correctly as depending upon one or other of these malformations.

The purpose of this paper is to attempt to show that malformations intermediate between the two extreme types already mentioned, present a definite clinical picture, are diagnosable by clinical methods alone, and are amenable in some degree to successful operative treatment. Its justification must be the successful diagnosis of four out of five cases seen within the last three years, in which the predicted malformation was subsequently found at operations undertaken deliberately for the purpose of rectifying it. In the fifth case no attempt at diagnosis was made, although significant physical signs were noted but not stressed beforehand in view of the fact that the patient had an acute abscess in her appendix.

A few facts of embryology must be recapitulated first, for the proper understanding of the significance of the clinical signs and symptoms present in these cases. The segment of gut involved—the mid-gut—is the whole portion of the alimentary tract which extends from the papillary portion of the duodenum to the junction of the middle and left thirds of the transverse colon. Alternately, it may be defined as all that portion of the alimentary tract which derives its blood supply from the superior mesenteric vessels. The small intestine is the first portion to be reduced within the abdominal cavity—the duodenum being followed by the jejunum and finally by the ileum—and during this stage it rotates from the coronal to the sagittal plane—a complete reversal of its position relative to the large gut in the umbilical sac. When the lowest part of the ileum enters the abdominal cavity only the cæcum and part of the ascending colon remain as the last contents of the umbilical sac, the rest of the ascending and transverse colon having been reduced *pari passu* with the small intestine. As the coils of small intestine begin to accumulate within the abdominal cavity a further process of rotation begins around the pedicle of the superior mesenteric vessels from the sagittal once more to the coronal plane, but in such a way that the coils of small intestine begin to pass behind the superior mesenteric vessels and that portion of the colon which has already entered the abdominal cavity. With the final completion of this phase of rotation, the small gut lies to the left side of the right colon and within the space bounded by the three segments of the colon, whilst its highest duodenal portion is crossed by the superior mesenteric vessels and by the colon in the region of the junction of its ascending and transverse portions.

The cæcum then sinks to the region of the right iliac fossa, the mesentery of the small intestine acquires a secondary fixation to the front of the bodies of the lumbar vertebræ, and the mesentery of the ascending colon disappears by fusion with the posterior parietes of the abdominal wall. If reduction and rotation are arrested during any of these stages then fixation and disappearance of the mesenteries does not occur, and it is for this reason that the title of "Congenital Malformation of the Mesentery" has been chosen for this paper. Whilst the clinical symptoms of these cases—more especially pain—seem to me to depend upon the failure of the primitive mesentery to undergo fusion and fixation, the clinical signs appear to depend upon the misplacements of the different segments of the midgut that must be associated inevitably.

The illuminating papers by Frazer and Robbins[1] and Norman Dott[2] should be studied by anyone interested in further embryological details.

With this prelude the clinical features common to all the cases can now be described.

1 Frazer, J. E., and Robbins, R. H., "On the Factors concerned in Causing Rotation of the Intestine in Man," *Journ. Anat.*, 1915, i.

2 Dott, Norman, "Anomalies of Intestinal Rotation," *Brit. Journ Surg.*, 1923, xi, 251.

Symptomatology.

The predominating symptom is pain, beginning as a rule in early infancy, and occurring intermittently in paroxysms, sometimes of great violence. When these pains have been studied in their clinical aspects of quality, intensity, duration, situation, conduction and radiation, and their modification by posture, food, and the action of the bowels, a composite mental picture of *unusualness* is at once formed. They are unlike anything that tradition and experience associate with the ordinary surgical disease of childhood, nor can they be dismissed summarily as forms of functional dyspepsia. Least of all have they any resemblance to any known lesion of the appendix. This *unusualness* will become more apparent when the details of the attacks of pain are described with each individual case.

Vomiting.

This was an entirely erratic symptom and was rare, except in Case V. It only occurred in Cases I and III occasionally; it tended not to be continuous, but appeared rather as a single reflex act of vomiting, in attacks when the pain was most intense, and the quantity and nature of the vomited material appeared to be determined entirely by what happened to be in the stomach at the time.

Abdominal Examination.

Clinical Signs (*a*).—In four cases a sense of "emptiness" of the right iliac fossa was a most arresting physical sign. This fossa is filled normally by large and small intestine, both of them being components of the mid-gut. Bearing in mind that malformations affect the segment of mid-gut only, it is clear that one or other of its components (it is not possible to dogmatize as to which) must be absent from the right iliac fossa, when, as it is easy to determine, this fossa is not normally filled. In Case III, I had stated beforehand that the emptiness of the right iliac fossa was due to the absence of the ascending colon, and this opinion proved to be quite wrong at the subsequent operation (Plate III). Emptiness of this fossa has been described as a sign of intussusception; it is very rarely so, because in this disease the fossa, as a rule, is more than generously filled by coils of over-distended intestine, which can be seen to occupy it during the course of the operations.

(*b*) An asymmetrical fullness is a fairly constant sign. It was noted in Cases II, III, IV and V. In Cases II and IV the whole of the colon was a left-sided viscus; in Case III the whole of the small intestine was lying external to the left colon and was a purely abdominal viscus, shut off entirely from the pelvis. In all these cases the whole of the left side only of the abdomen was obviously swollen on inspection; palpation confirmed an abnormal fullness without any evidence of tumour formation. In Case V the whole of the upper half of the abdomen was swollen; below the umbilicus it appeared abruptly flat.

X-ray Examination.

Radiography applied with the special purpose of confirming the clinical diagnosis of congenital malformation proved to be disappointing on the whole. In Case I a high cæcum was revealed; but the intimate relationship of the duodenum and terminal part of the ileum enclosed by a band of omentum could not, of course, be shown. No help was obtained from pictures of the other cases; but it was not

realized until after the operation on Case III that the best way to obtain information as to the relative positions of the small and large intestines is to give a barium meal by the mouth, and, when this has been seen on the screen to be giving a good definition of the small intestine, to administer a barium enema and thus obtain a composite picture. This method was adopted for Case III two months after operation, when a perfect picture was obtained showing that the small intestine now occupied its normal position between the three segments of the large bowel.

Details of Cases.

(I) N. B., female, aged 6½ years, was first seen February 25, 1925, with Dr. H. Waller.

History.—She had a "weak digestion" from infancy, and had been under continuous treatment for this by a consulting physician for the past three years. The attacks of pain were of a violent and paroxysmal nature, experienced in the region of the right hypochondrium, and might last for only a few minutes or for a couple of hours. In the worst attacks the child would throw herself upon the floor and scream. They were liable to come on peculiarly during exertion, most frequently when she was running about playing out of doors, or towards the end of the day when she was having a final romp in the nursery before going to bed. They had no time-relationship to food, and appeared to be quite unrelated to regularity or irregularity in the action of the bowels. They had been present from early childhood. The parents had been advised now that the appendix ought to be removed.

On Examination.—The singular emptiness of the right iliac fossa was noted, but nothing else abnormal was detected. Skiagrams seemed to show that the cæcum was under the liver. Taking these findings in conjunction with the details of her attacks of pain, a diagnosis of "congenital malformation of the mesentery" was made and operation advised. The parents refused to agree to this, and as they were met with a refusal to remove the appendix, the child was placed under the care of another dietetic specialist for a further period of fifteen months. At the end of this time her sufferings had been entirely unaffected, and consent was given now to the performance of the operation advised originally (Plate I).

Operation.—Laparotomy was performed June 9, 1926. When the abdomen had been opened by reflecting the right rectus outwards, the whole of the small intestine was found to be on the right side. The duodenum was a straight tube with the terminal portion of the ileum close alongside and parallel to it. These two portions of the small intestine were bound together by a stout anomalous band of omentum which completely encircled them and at the same time enclosed the whole of the mesentery which contained the superior mesenteric vessels. The ileum joined the cæcum immediately below the greater curvature of the pyloric end of the stomach; from the cæcum the large intestine passed directly to the left, so that the ascending and transverse colons appeared to be represented by a short segment of colon between it and the splenic flexure. The colon on the left side was voluminous. A small and entirely healthy-looking appendix was removed. The anomalous band of omentum was resected freely so as to liberate entirely the pedicle formed by duodenum, superior mesenteric vessels, and ileum which was constricted by it. A congenital foramen (not shown in the sketch) in the mesentery of the small intestine was closed. Nothing else abnormal was found.

Results.—The child had an easy recovery, and up to the present time (May, 1928) has remained perfectly well without any recurrence of her former troubles. It would appear from a survey of all the details of the case that the constricting band of omentum was the chief factor in producing the attacks of (violent) abdominal pain, either by obstructing at intervals the lumen of one or other of the enclosed pieces of gut, or by forming a pedicle so narrow that torsion occurred in the direction of a volvulus which was prevented from becoming complete by the broad attachment of the omentum to the stomach. In favour of the latter view the liability of the attacks to be precipitated by active exercise is suggestive.

(II) R. F., female, aged 12 years and 4 months, seen in conjunction with Dr. R. S. Frew.

History.—All her life she had had a "delicate inside," and at the ages of 4, 6, and 8 years respectively, had had attacks of severe abdominal pain lasting for two days, and associated

with the passage of blood and mucus from the bowel, so that intussusception had been suspected. Her own description indicated a gnawing pain over the area of the stomach entirely to the left of the middle line. This pain always began a few moments after swallowing the first few mouthfuls of a meal, and increased in intensity as the meal progressed, reaching its maximum at the end of the meal. From this point it began to die away gradually and as a rule had just disappeared before the next meal was due. It was always worse during the two large meals of the day, i.e., breakfast and dinner. Posture had no influence upon it except that the patient could endure an inevitable evil more comfortably when she was lying down, and no regulation of dietary or action of the bowels had any effect upon it. She had been brought up on careful diet and on drugs to control the action of the bowel daily, but at no time did she obtain any prolonged freedom from her gastric pains. She was sent up from her school at the seaside to London with a diagnosis of acute appendicitis, and I first saw her on April 22, 1925.

On Examination.—The right iliac fossa was obviously empty, and there was no tenderness anywhere. The left side of the abdomen was full in its lower half only. In view of the child's own excellent description of her pain, the presence of these signs appeared to justify the diagnosis of "congenital mesenteric malformation"; and the parents consented to an operation to investigate this condition. There were no signs at all of any acute illness, so that it was arranged for convenience that the operation should be done on May 6, 1925 (Plate II).

Operation.—The right rectus was turned outwards and the abdomen opened. The small intestine occupied the right side of the abdominal cavity, and the whole of the colon was on the left side; but, unlike that in the previous case, the cæcum was lying on the left side of the floor of the pelvis, where the ileum joined it. The ascending colon made a vertical ascent to reach the greater curvature of the stomach to the left of the middle line; from this point to the splenic flexure the transverse colon was represented by a short segment of gut only. The ascending colon was enclosed by the same primitive layer of mesentery as the small intestine; no secondary fixation had occurred anywhere, so that the whole weight of the primitive mid-gut was supported by the stomach. The condition was one of total reduction of the mid-gut with arrest of subsequent rotation and fixation. A small and very innocent-looking appendix was removed, and the abdomen closed with the feeling that no improvement could follow.

Results.—The attacks of gastric pain recurred ten days later, and continued at frequent intervals throughout the following year. Treatment was devised after operation with a view to taking the weight of the colon off the stomach as much as possible. A supporting belt was ordered, which was put on when the child was "upside down," a diet devised to leave the smallest amount of debris, and mild aperients and paraffin were prescribed to secure a a rather frequent emptying of the intestine. During the present year there has been some improvement in her condition.

(III) L. B., male, aged 10 years, was admitted to the Hampstead General Hospital on January 28, 1927, under the care of my colleague, Dr. Scott-Pinchin, who kindly asked me to see the patient with him. Under observation in the ward an attack of left-sided abdominal pain, with distention of the abdomen and a passage alternately of "pipe-stem" and enormous motions, had been noted.

History.—The full sequence of events which had been present from early childhood was described by the mother who had taken him to many hospitals in London for treatment of his "indigestion"; and on two occasions had refused her permission for the removal of the appendix. A pain like "a bad stomach-ache" was experienced on the left side of the abdomen spreading well out to the left flank. A constant series of changes observed by the mother heralded and ushered out each attack: daily motions became small and pipe-like, then there was complete constipation for a few days, then a slow symmetrical swelling of the whole abdomen began which was followed after a varying period by the attack of pain. This might last for an hour or two or for most of the day, and was always relieved by the spontaneous passage of several enormous, formed motions. The attacks of pain were not affected by posture, or by changes in diet, or by drugs, which had been administered for several years.

84 Waugh: *Clinical Aspect of Congenital Mesenteric Malformation*

On Examination.—He was first seen by the writer on March 21, 1927, when the left side of the abdomen was noticed to be obviously swollen; it was full on palpation, without any evidence of tumour formation, and the right iliac fossa felt abnormally empty. A diagnosis of "congenital mesenteric malformation" was made, with which Dr. Scott-Pinchin agreed, and operation was decided upon after further X-ray examination. The radiograms were not helpful, and it was only after operation upon this case that the right method of procedure was worked out (Plate III).

Operation.—Laparotomy was performed on April 1, 1927. The left rectus was turned outwards and the abdomen opened. It was then found that the first part of the jejunum and the terminal part of the ileum converged to a narrow pedicle crossing and tying down the mid-point of the left colon, external to which the whole of the rest of the small intestine was lying. This latter part was confined entirely to the abdominal cavity and left iliac fossa, since all access to the true pelvis was cut off by the iliac and pelvic colon. Where the mesentery of the small intestine crossed the left colon it was tied to it by a stout band of primitive undifferentiated embryonic tissue and the colon was flattened, and only when this had been cut through was it possible to reduce the small intestine to its normal pelvic and abdominal situation bounded by the three segments of the colon. It settled down here quite easily without any impediment to its circulation, or any tendency to return to its abnormal position. A simple healthy appendix was then removed and the abdomen closed (Plate IV).

Results.—The boy has remained quite well up to the present time, and has shown no tendency to a recurrence of the alternate small and large motions. Two months after the operation a skiagram of the abdomen was taken with the improved procedure, and the small intestine was clearly outlined lying in its normal position in relation to the colon. The clinical features of the case noted beforehand became easily understandable in the light of what was found at operation.

(IV) E. H., female, aged 21, was seen with Dr. E. A. Seymour, on January 26, 1927. The symptoms of the attack of acute appendicitis from which she had just recovered need no description. They were confirmed at the subsequent operation, when an appendix full of pus with recent lymph around it was removed.

On Examination.—The emptiness of the right iliac fossa was noted, as well as a slight fullness of the abdomen on the left side. No deductions were made from these very important observations, which were overshadowed entirely by a description of the symptoms of an acute illness from which the patient had just recovered successfully.

Operation.—Laparotomy was performed on February 2, 1927. The lower half of the right rectus was turned outwards and the abdomen opened. The position of the viscera was identical with their position in Case II (Plate II). One difference—possibly of importance in view of the absence of a history of prolonged "indigestion"—was noted in this case. The ascending colon had acquired, over its whole length, a secondary attachment and firm fixation to the left side of the bodies of the lumbar vertebræ, and was not contained within the fold of primitive mesentery common to it and the small intestine; in Case II the ascending colon had not formed any such attachment and was completely mobile.

Result.—The patient has been quite well since the date of operation when the appendix was removed.

(V) A. V. R., male, aged 7½ years, was admitted to the Hampstead General Hospital on October 6, 1927, under the care of my colleague, Dr. Scott-Pinchin, who kindly asked me to see the case with him.

History.—From early infancy the patient had suffered from attacks of severe abdominal pain and vomiting. The pain was very violent in character, and was experienced by the patient in the mid-epigastric region. The first attack occurred before he was 2 months old, and was accompanied by continuous vomiting, which became biliary in character. By the time he was 7 years old it was easy to recognize that these attacks were characteristic of an obstructive lesion high up in the alimentary tract. The vomiting quite often occurred independently of the pain, was copious to begin with, continuous, and consisted ultimately of bile. At the age of 2 months he was admitted to a children's hospital in London, and was

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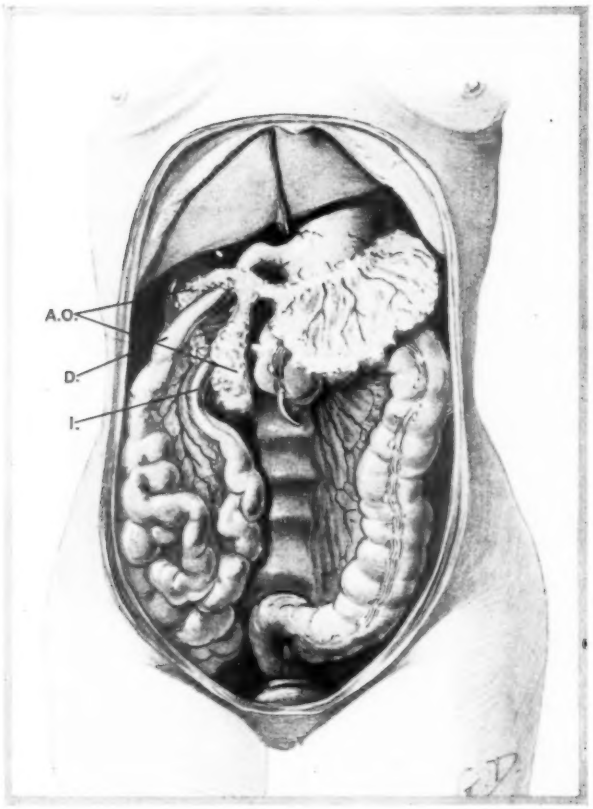
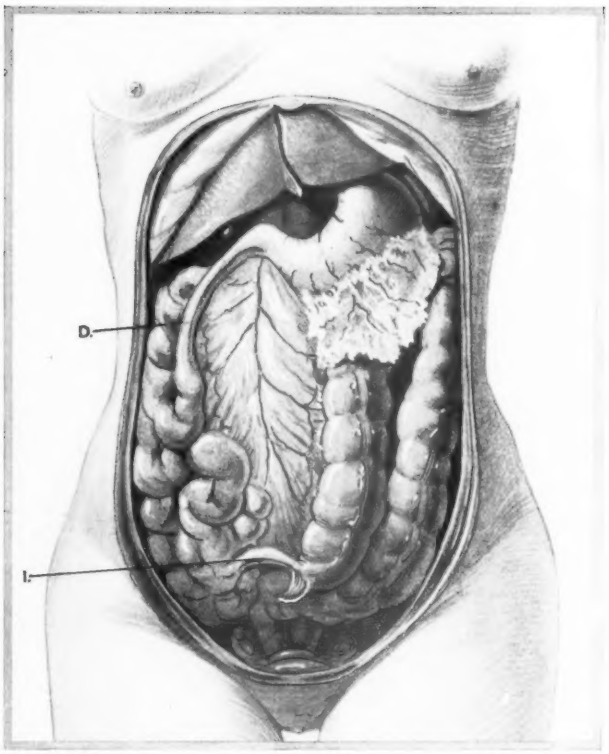
PLATE I.[1]



Case 1.—*AO*, Abnormal band of omentum encircling: *D*, Duodenum: *I*, Terminal part of ileum; and the root of the mesentery containing the superior mesenteric vessels. The whole of the small intestine is at the right side of the abdominal cavity.

[1] This illustration and the five following plates are reproduced by permission from the *British Journal of Surgery*.

PLATE II.



Cases 2 *and* 4.—*D*, Duodenum: *I*, Terminal portion of the ileum. The whole of the small intestine is at the right side, and the whole of the large intestine on the left side of the abdominal cavity. In *Case* 4 the whole of the mesentery of the ascending colon was firmly attached to the left side of the bodies of the lumbar vertebræ. In *Case* 2 no fixation of the common mesentery of the whole loop of mid-gut had occurred.

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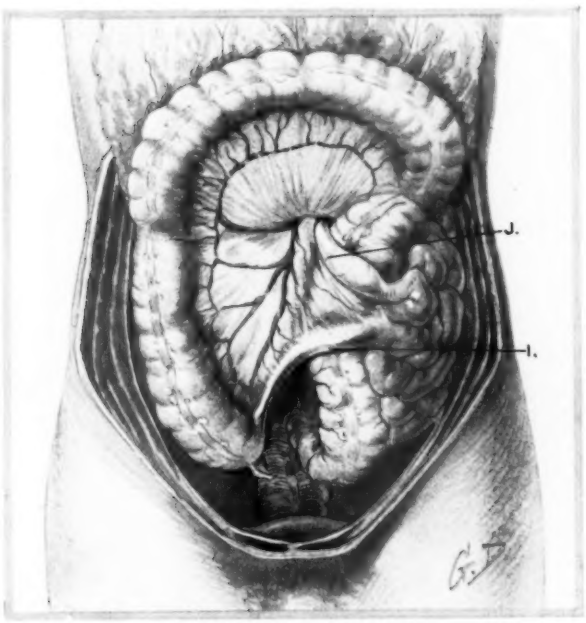
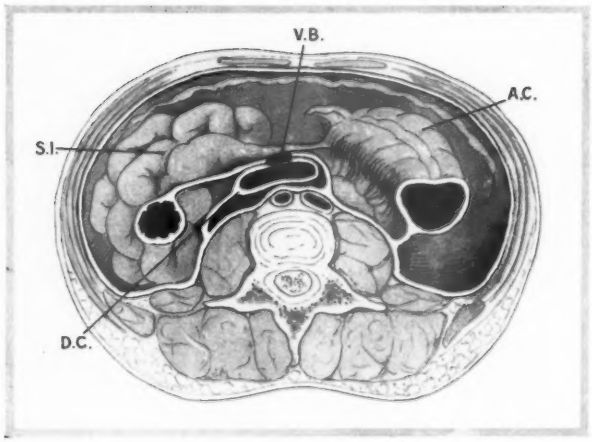
PLATE III.



Case 3.—*J*, First part of jejunum, and *I*, Terminal part of ileum, forming a pedicle crossing and tying down the descending colon, external to which the whole of the rest of the small intestine was lying on the left side of the abdominal cavity.

PLATE IV.



Case 3.—Diagram of transverse section of abdominal cavity to show the descending colon flattened by the pedicle of the small intestine which crossed it, and the vestigial band joining the mesentery of the small intestine to the anterior surface of the descending colon. This band had to be cut through before the reduction of the small intestine to its normal position could be effected. *DC*, Descending colon: *SI*, Small intestine: *VB*, Vestigial band: *AC*, Ascending colon.

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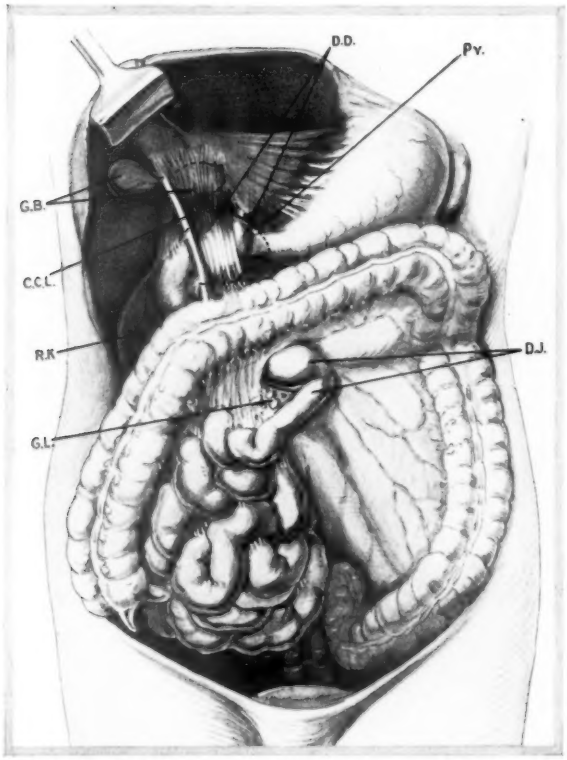
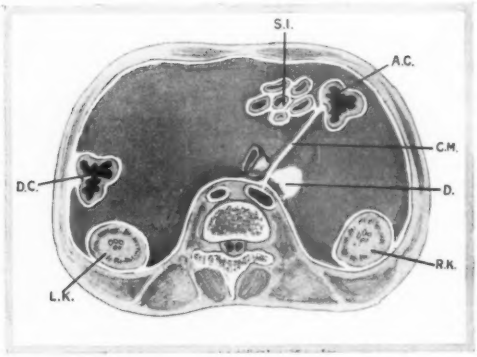
PLATE V.



Case 5.—*GB*, Gall-bladder in dotted outline beneath cysticocolic ligament; *CCL*, Cysticocolic ligament with thickened free edge; *RK*, Right kidney; *DD*, Dilated portion of duodenum in dotted outline beneath cysticocolic ligament; *PY*, Pylorus. The ascending colon and small intestine upon their common mesentery have been turned to the right to display the duodenojejunal junction (*DJ*). This shows an abrupt angle containing enlarged lymphatic glands (*GL*), and the terminal portion of duodenum dilated, in contrast with the lumen of the first part of the jejunum. The much larger masses of glands along both borders of the whole of the duodenum are not shown in the sketch.

PLATE VI.



Case 5.—Diagrammatic cross-section to show the common mesentery of the small intestine and colon and the relation of the vascular pedicle to the horizontal portion of the duodenum. *SI*, Small intestine; *AC*, Ascending colon; *CM*, Common mesentery; *D*, Duodenum; *RK*, Right kidney; *LK*, Left kidney; *DC*, Descending colon.

under treatment for one month as an in-patient. He was admitted during the following seven years to four other hospitals, but the attacks had continued unaffected by any form of treatment until his admission to the Hampstead General Hospital.

On Examination.—When first seen on October 6, 1927, he was a frail, sickly-looking boy, weighing 2 st. 9½ lb. The whole of the upper half of the abdomen above the trans-umbilical plane was distended symmetrically, and the lower half appeared flat by contrast. No mass formation was detectable, and visible peristalsis was noted high up in the right hypochondrium. This was clearly intestinal—and not gastric—peristalsis. Radiograms of barium meals showed that retention occurred in an otherwise normal stomach for more than six hours. A second series of radiograms was taken after the administration of atropine, and these excluded pylorospasm as a possible cause of the trouble: a persistent organic obstruction of the pylorus was excluded by the presence of bile in the vomit; whilst congenital atresia of the duodenum or jejunum seemed unlikely in a patient who had reached the age of 7½ years. A "congenital mesenteric malformation" was therefore diagnosed, which, acting as an extrinsic force, produced intermittent obstruction high up in the alimentary tract (Plate V).

Operation.—Laparotomy was performed on October 27, 1927, and the upper half of the right rectus was turned outwards. The stomach appeared to be quite normal, and the pylorus was soft and devoid of irregularity, and admitted the tip of an index finger easily. An unusual form of cysticocolic membrane was found, whose free edge was thickened by a stout band. This band began at the transverse fissure of the liver and passed downwards across the gall-bladder, which it constricted into a figure of ∞. It then continued over the anterior surface of the second part of the duodenum to end on the hepatic flexure of the colon. The duodenum was tightly constricted at this crossing and widely ballooned above it. The whole of the membrane was cut away up to the foramen of Winslow, with immediate relief to the constrictions of both gall-bladder and duodenum. It was then noted that the whole of the rest of the duodenum appeared to have a slightly larger lumen than the jejunum, and that the first part of the jejunum was bent at an acute angle to the terminal part of the duodenum. This appeared to be due to abnormal mesenteric attachments of the rest of the small intestine and the right colon. These viscera were contained in a common mesentery, whose sole line of attachment posteriorly was the usual one of the small intestine, but only to the body of the second lumbar vertebra. Consequently the whole weight of the colon and small intestine combined fell upon the vascular pedicle crossing the horizontal portion of the duodenum and upon the other fixed point of the gut, i.e., the duodenojejunal flexure. As, however, the dilatation of the duodenum that might be attributed to these two factors was much less than the gross dilatation above the crossing of the cysticocolic ligament, it was decided to do nothing more for the present. If the progress of the patient does not continue in a satisfactory manner, then a relief of the strain on the duodenum, either by fixation of the ascending colon or by the performance of a duodenojejunostomy, as advised by Wilkie, will have to be considered (Plate VI).

Another feature of great interest was the presence of enlarged lymphatic glands distributed along both borders of the duodenum and forming large clusters between the layers of the gastro-hepatic omentum, over the head of the pancreas, and at the duodeno-jejunal junction. There were none in the whole of the rest of the mesentery, so that this distribution appeared to suggest that a simple infection had occurred by adsorption from the retained contents of the duodenum. One gland was removed for microscopical investigation, and the pathological report was to the effect that the enlargement was due to simple inflammatory changes and that there were no signs of tuberculosis. A small healthy-looking appendix was removed at the end of the operation.

Results.—The general condition of the patient has improved very much since the operation, from which he had a very easy recovery. Another series of skiagrams taken on November 17, 1927, shows that the stomach now gets rid of a barium meal at the end of three hours—that is, just twice as quickly as before the operation. He has had no further attacks of pain, and his weight had increased from 2 st. 9 lb. to 3 st. 2 lb. on April 15, 1928.

86 Paterson : *Bilateral Hernia Diaphragmatica*

CASES.

Bilateral Hernia Diaphragmatica.

By DONALD PATERSON, M.B.

M. J., A GIRL, aged seven years and one month, admitted to Children's Ward, Westminster Hospital, on April 16, 1928. Complaint: Swelling in the abdomen and failure to thrive since the age of 9 months. One of eight children; the others all healthy. For the last year she has suffered from attacks of indigestion.

FIG. 1.—Note the short stature (for a child aged 7), the lax stance, and prominent abdomen.

On Examination : She was much undersized, looking like a child aged 5 (fig. 1) but her mentality was quite normal. A large mass, felt in the right iliac region, proved to be the liver. This could be lifted up and replaced beneath the costal margin on the

right side in its normal position, but it immediately fell forward and rolled back into the right iliac region when left alone. The chest was hyper-resonant all over, there being no area of cardiac dullness. The heart sounds were most indistinct in the cardiac region and were heard best at the angle of the scapula posteriorly. The blood-count showed the hæmoglobin to be 116 per cent., red cel's 5,800,000, white

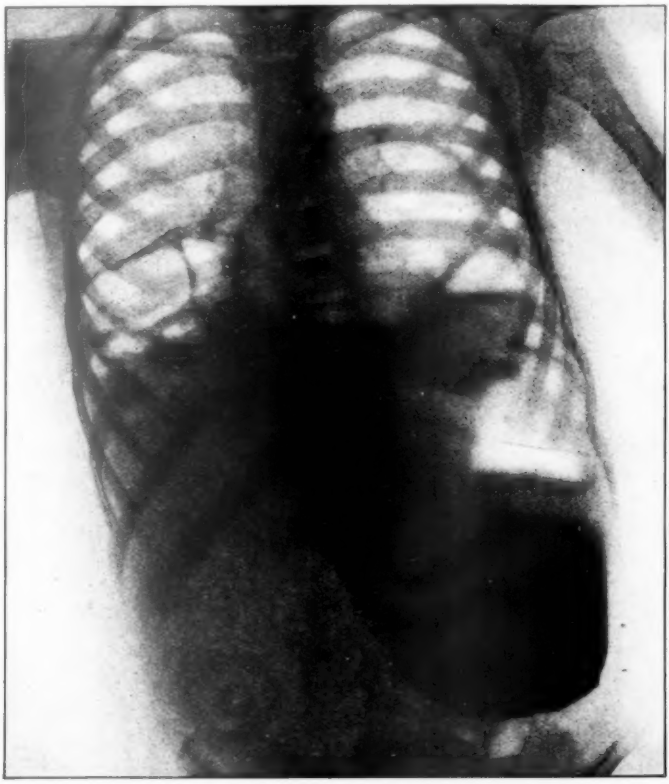


FIG. 2.—Bismuth is seen in the lower pouch of a much dilated stomach. Above the level of the diaphragm the upper pouch is seen in outline filled with air. When inverted, the bismuth ran into this upper pouch.

cells and differential count normal. The air entry was poor over the front of the chest and good posteriorly. On examination of the abdomen, peristaltic waves could be seen, which were thought to be due to some intestinal obstruction. With a bismuth meal the stomach was seen to be enlarged and bilocular, the upper portion lying above the diaphragm on the left side of the chest. When the child was inverted, the bismuth flowed from the lower compartment to the upper one, demonstrating this fact fully. A few hours later the bismuth could be seen in the small intestine, now lying in the right side of the chest, filling almost the whole of the right chest (fig. 2). This suggests a bilateral congenital deformity of the diaphragm allowing the abdominal contents to herniate through.

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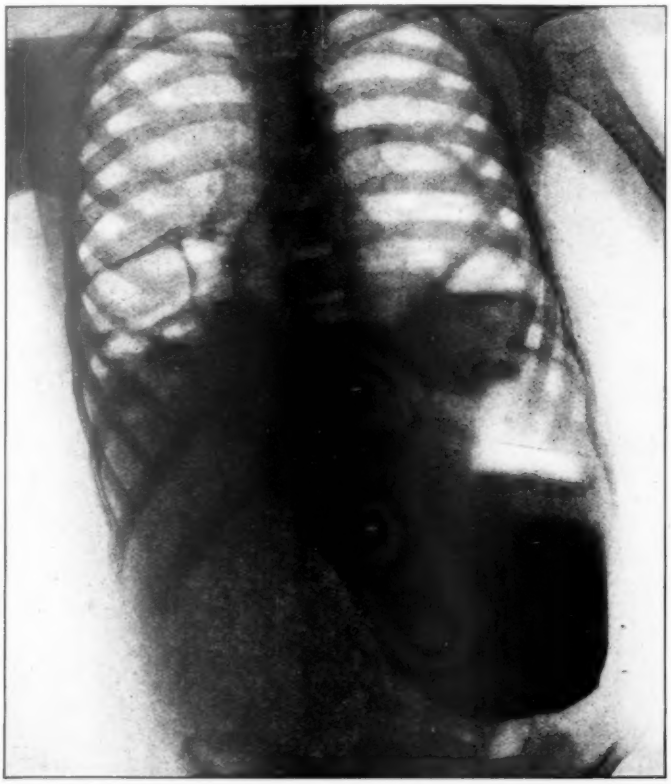

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88 Paterson : *Bilateral Hernia Diaphragmatica*

In a recent paper by Hedblom, this author states that in a third of all these cases the hernia strangulates sooner or later. When operated upon there was

FIG 3.—The bismuth is seen in the small intestine in the right side of the thorax. A small fragment of bismuth remains in the stomach in left thorax.

a 53 per cent. mortality among the obstructed cases and only a 23 per cent. mortality among the non-obstructed. Some beautiful skiagrams of the condition were shown by Carman and Fineman in their article in *Radiology*.

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Mr. G. E. WAUGH (President) said that if an opaque stomach-tube was passed it could be ascertained whether there were two openings or only one. From the picture there seemed to be some doubt. Without knowing on which side the opening was, or whether there was an opening on both sides, the question of surgical treatment was necessarily difficult. When that point was cleared up, it would be possible to say whether or not the child should have a thoracic operation.

Epidermolysis Bullosa Hereditaria, with Three Illustrative Cases.

By RAYMOND GREENE, L.R.C.P., M.R.C.S. (introduced by Dr. DONALD PATERSON).

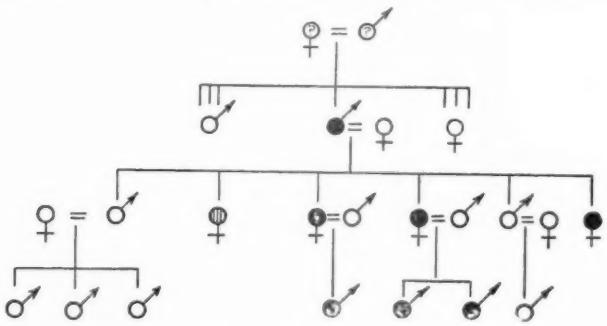
EPIDERMOLYSIS BULLOSA HEREDITARIA is a rare hereditary skin disease, characterized by an extreme sensitiveness of the skin to the slightest trauma. The least conspicuous irritation, knock, or pinch, immediately gives rise to intense irritation, followed by the appearance of a bulla. Two types have been distinguished by Luithlen. In the first the blebs contain a clear fluid, and heal rapidly without scarring. In the second, to which my cases belong, the blebs contain blood-stained fluid, and heal with much scarring and permanent pigmentation. The second type shows varying degrees of nail dystrophy, from slight brittleness to marked deformity, or even absence. It is often associated with epidermal cysts, which are absent in my cases. It may affect the mucous membrane as well as the skin, and one isolated case reported by Klausner developed epithelioma of the buccal mucous membrane at the age of twenty-five. Neither type primarily affects the general health, but patients are subject to septic complications, which tend to shorten their lives. Some observers have stated that the condition improves with advancing years, but Augagneur published a description of a patient aged 55, and the grandfather of these two children is aged over 60. The first case reported was by Tilbury Fox in 1879. Since then the number of reported cases has been small.

The pathology of the condition needs further investigation. Crocker ascribes it to an excessive irritability of the vasomotor nerves of the skin, by which the natural protective mechanism is "overdone." Elliot demonstrated a raising up of the greater part of the rete, and showed degenerative changes in the rete in apparently normal skin. Ergmann and Mook found in their cases a distinct diminution of the elastic tissue element.

The family history of my cases has been worked out to three generations. In the accompanying genealogical table sufferers are shown in black, those free from the disease in white.

90 Greene : *Epidermolysis Bullosa Hereditaria; Illustrative Cases*

The first generation consisted of two who are said to have been free. The second generation shows a male sufferer and an unknown number of persons of both sexes said to be free. The male sufferer produced two free males, three affected females, and one female who is said to be hermaphrodite, and to be affected by "another skin disease." The free males produced free offspring. One affected female has no children. The remaining two females have produced families of affected males. The tree shows an interesting alternation of sexes. It is unfortunate that the fourth generation consists entirely of males. I have not yet had the opportunity of examining other genealogical trees in the literature.



The mother and her two children in the cases shown exhibit the typical lesions of the disease, the primary hæmorrhagic bullæ and the pigmented papyraceous scars of the healed condition. The majority of the bullæ and scars are in the neighbourhood of joints.

FIG. 1.—Showing fully developed hæmorrhagic bullæ over the elbow-joints.

Dr. Braxton Hicks has made the following pathological reports concerning the two children :—

(1) *Blood-counts.*—

	John	Kenneth
Hæmoglobin...	75 per cent. ...	100 per cent.
Red blood-corpuscles	6,110,000 ...	5,000,000
Colour-index	0·6 ...	1·0
White blood-corpuscles ...	15,800 ...	9,400
Polymorphonuclears	55·5 per cent. ...	50 per cent.
Monocytes	1·0 per cent. ..	4 per cent.
Lymphocytes	43·0 per cent. ...	44 per cent.

(2) *Contents of Bullæ.*—Smears prepared from the fluid show the presence of red blood-corpuscles and polymorphonuclear leucocytes in fair numbers. There is no evidence of the presence of fibrin clot. Chemically, the fluid contains albumin and globulin, but no fibrinogen. The total protein content is 5·8 per cent., of which 72 per cent. is albumin. No abnormality in the chloride content is detected. The amount available was too small to determine the amount of calcium present. No organisms were found (Gram and Loeffler). Cultures in agar, blood-agar and broth were sterile in forty-eight hours.

(3) *Wassermann reaction* negative in both brothers.

FIG. 2.—Showing (i) nail dystrophy; (ii) a hæmorrhagic bulla on the right knee; (iii) papyraceous scarring of both knees.

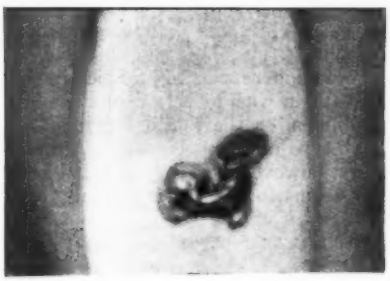


FIG. 3.—Showing a typical bulla.

There is a strong family tradition that sugar increases the severity of the symptoms. During his time in the children's ward at Westminster, the younger brother refused sweet food, a notable point in a child of four. I propose in the near future to inquire further into this tradition, and also, if the necessary permission can be obtained, into the histology of the lesions.

I have to thank Dr. S. E. Dore for his permission to show the elder child and Dr. Donald Paterson for his permission to show the younger.

Congenital Anæmia with Jaundice.

By HAZEL CHODAK GREGORY, M.D.

PATIENT, a male child aged twenty-four days, born April 24, 1928, after normal labour of sixteen hours' duration. Birth weight 8 lb. 14 oz. Child was reported to be white and flaccid at birth but soon cried and appeared normal. Jaundice was first noticed ten hours after birth and the urine stained the napkins. Child taking breast feeds well. On April 29 he vomited after one of his feeds and has vomited occasionally after feeds since. Vomit not stained by bile.

Family History.—Parents alive and healthy. Patient is fourth child. One stillbirth. Mother healthy during pregnancy. Mother's Wassermann reaction negative. Father's not obtained. No history of jaundice in family.

General Condition.—When seen May 1, patient was deeply jaundiced. Stools yellow. Mucous membranes bile-stained. Umbilicus healthy. Liver palpable one finger's breadth below the costal margin. *Spleen:* firm and much enlarged.

Admitted to Ward, May 10.—Jaundice was marked but colour distinctly pale. Purpuric patches on forehead at roots of hair. Mucous membranes pale. Liver two fingers' breadth below costal margin. Spleen still large. Stools healthy, well-coloured. Urine: dark, bile-stained.

Pathological Report.—Urine (May 10, 1928): acid; no albumin, acetone or sugar; no urobilin; bile present. Blood-count (May 11, 1928): red corpuscles, 1,605,000 per c.mm. White corpuscles, 13,200 per c.mm. Hæmoglobin, about 30 per cent. Red cells show polychromasia, and many nucleated forms are present. Polymorphonuclear cells, 15 per cent.; large lymphocytes, 7 per cent.; small lymphocytes, 45 per cent.; eosinophil, 1 per cent.; hyaline, 11 per cent.; myelocytes, 2 per cent.; plasma cells, 4 per cent.; atypical mononuclear cells, 15 per cent.; by oxidase method of staining, 14 per cent. polymorphonuclears and 11 per cent. cells contain oxidase granules. In counting 100 cells, 23 normoblasts and 13 megaloblasts were seen. *Hæmolysis* of red blood-corpuscles begins in 0·34 per cent. saline—complete in 0·3 per cent., showing an increased resistance. *Ratio of platelets to red cells,* $\frac{1}{26}$. Platelets, 61,500 per c.mm. Wassermann reaction negative.

Progress.—Since admission child has had one small epistaxis. Taking breast feeds well. Occasional vomit.

This is not one of the commoner forms of jaundice in the new-born child, and the symptoms and signs do not fit in with icterus neonatorum, congenital obliteration of the bile-ducts, septic umbilicus, or the familial type of acholuric jaundice. They seem to fit in best with the type of jaundice with anæmia described by Buchan and Comrie.[1] Their cases were, however, generally familial and the babies were actually born with jaundice, which seems to have been generally of the obstructive variety.

Postscript.—Since the case was reported a streptococcus has been found on two occasions in the infant's urine. This organism is being investigated.

Dr. KINGSTON BARTON said he believed that in the majority of these cases of jaundice occurring soon after birth the patients had hæmorrhage from the bowel within a few days, and died within a short time, i.e., about ten days. It would be interesting to know what was the significance of a combination of anæmia and jaundice. This present case was unusual, for though there had been epistaxis once, there was no evidence of hæmorrhage from the bowel. The prognosis did not seem to be favourable.

1 A. Hill Buchan and J. D. Comrie, *Journ. of Path. and Bacterio.*, xiii, 398.

Section of Obstetrics and Gynæcology.

President—Mr. Comyns Berkeley, M.Ch.

[*June* 15, 1928.]

A Case of Dysmenorrhœa due to Calcification of the Ovary.

By A. Louise McIlroy, M.D.

The patient, a single woman, aged 24, engaged in office work, was sent to me a year ago on account of dysmenorrhœa of twelve months' duration. Her medical attendant had diagnosed a hard rounded swelling in the pelvis. The history was as follows: Menstruation began at 14 years, was of twenty-four to twenty-eight day type, and lasted six days, the loss being normal in amount. She had always been healthy until the past year when she had complained of a severe dragging pain in the left iliac region which began one week before the onset of the menstrual period and lasted about two weeks. The pain was severe when she was sitting still and sometimes caused her to faint. For the past three months the menstrual loss had slightly increased and dyspepsia had been present. Micturition had been more frequent and slightly painful. The bowels were regular. No vaginal discharge was present. On abdominal examination there was tenderness and rigidity in the left iliac region but no swelling was felt. On rectal examination a hard swelling, not attached to the uterus, was felt in the pelvis, and a calcified ovary or pedunculated fibroid was diagnosed. No vaginal examination was made.

The abdomen was opened and a nodular, non-adherent, stony tumour about the size of an ordinary pear was found on the right side. This was the right ovary. The uterus and other ovary were normal. The tumour was removed and the patient made an uneventful recovery, the wound having healed on the fifteenth day.

The tumour weighed 6 oz. and was somewhat nodular in shape with some venous tracings over its surface. It was pinkish in colour with yellow patches in places. Its consistence resembled that of a stone. Part of the Fallopian tube which formed the pedicle was attached to the tumour.

Professor Dible, in his report, stated that the tumour was a completely calcified ovary which could not be cut. X-ray examination revealed no further information as to its structure.

The chief interest of the specimen consists in its rarity and its occurrence in an otherwise perfectly healthy young woman.

In a recent report from the patient, she states that she has been perfectly well since the operation and that her periods have been regular and free from pain.

Abdominal Carcinoma with Vulvo-Vaginal Metastases.

By Clifford White, F.R.C.S.

I am drawing attention to this condition because it is a rare one, and because it is clinically of importance to recognize that a metastasis in the lowest portion of the vagina may occur in a case of carcinoma of the uterus or ovary.

It is well known that in cases of carcinoma of the cervix nodules of growth may extend down to the vulva, although there may be areas of apparently healthy vaginal mucosa separating them from the cervix; again, in cases of malignant ovarian cysts that have eroded through the pouch of Douglas there may be a similar condition of the vagina. But in the cases to which I am now referring there was no question of any gross involvement of the vaginal vault, and in two cases the only metastatic deposit that could be detected on examination was a single one at the junction of the vagina and the vulva.

I saw the first case in December, 1926, when I was consulted by Mrs. J., aged 56, who had had seven children. The menopause had occurred eight years before, but she had had a bloodstained discharge for four months previously to my seeing her. On examination, I found a tumour in the lower abdomen and what I took to be a small papilloma in the vulva, below the urethra. The patient was suffering from diabetes of a serious type, and I had this treated by a physician and did not open the abdomen until two or three weeks afterwards. I then found the right ovary 10 cm. in diameter and the left half that size. Both were adherent to the uterus and also to the intestine, especially on the right side. With some difficulty I removed both tumours and the body of the uterus.

There was an irremovable nodule on the bladder wall, but the pouch of Douglas was unaffected. No other focus of growth was found in the abdominal cavity.

I also removed the nodule on the anterior vaginal wall. It was much larger than when I had first examined the patient and measured 3 cm. long and 2 cm. broad. It was raised and purple in colour. On examining the lower part of the vagina more fully, I found a smaller similar growth on the posterior wall and several quite small ones, all on the lower part of the vagina.

On section, the cysts and the vaginal growth showed adeno-carcinoma; the vaginal growth being identical in structure with the ovarian carcinoma.

The patient died about three months later.

In the second case I did not operate, and therefore cannot prove microscopically that it was of the same type as in the first case, but the clinical resemblance is so close that I am recording it.

The patient was a multipara, aged 70, who had abdominal pain and swelling and also a bloody vaginal discharge. I saw her in August, 1927, and found a large semi-solid abdominal tumour with free intraperitoneal fluid. On pelvic examination, the vaginal vault did not strike me as abnormal, but just inside the vulva there was a single nodule three and a half cm. in diameter. It was soft to touch but not ulcerated, its colour was mauve and it resembled in every way the growth found at operation in the first case. The patient was in very bad general condition, and all that could be done was to give morphia. She died a few days later, and although I asked the doctor to try to obtain a small portion of the vaginal growth so that I might have a section made, he was unable to do so, and therefore I cannot prove that the vaginal growth was an adeno-carcinoma, but, judging from its exact resemblance to the growths in the other cases, I believe that it was an adeno-carcinoma secondary to the ovarian carcinoma.

The third case is that of a multipara, aged about 40. In May, 1927, she had

multiple uterine fibroids causing menorrhagia, and an abdominal panhysterectomy was performed by Professor Lowry, in Belfast. On examining the specimen after the operation, a small growth was found in the body of the uterus, which, on section, proved to be an adeno-carcinoma.

The patient had an uninterrupted recovery and remained well until February, 1928. She then noticed a trace of pinkish discharge and saw me on March 29. I found a pale, purple, soft raised growth on the left of the urethral orifice. The appearance was identical with that in the first two cases.

No other nodules could be found, even after a thorough examination; there was no thickening of the cellular tissue or of the vaginal vault.

I referred her to Professor Lowry again, and he excised the nodule of growth and the inguinal glands and treated her with deep X-rays. He kindly sent me sections of the growth, and they show typical adeno-carcinoma such as one sees constantly in carcinoma of the body of the uterus.

I am unable to explain how a metastasis from a tumour in the lower abdomen reaches the vulva. It would appear to occur against the direction of the blood- and lymph-streams. Such metastases have been recorded in cases of chorion-epitheliomata, and it has been suggested that they result from a retrograde permeation of the veins. This in itself is difficult to conceive, and further, if malignant tissue once gets into the blood-stream, a generalized dissemination all over the body may be expected to occur (as in some of the recorded cases of chorion-epithelioma), not a single vulvo-vaginal growth with no evidence of pulmonary or other deposits. Cases of vulval growths after carcinoma of the body of the uterus are recorded in the literature, but, in some of these, the uterus had been removed by an extended vaginal hysterectomy with a Schuchardt's incision, so that they are rather examples of implantation such as are seen in perineal endometriomata.

Cullen ("Carcinoma of the Uterus," p. 415) records one somewhat similar case, discusses the possibility of a backward flow of lymph, and gives references to other cases.

Frank ("Gynæcological and Obstetric Pathology," 1922, p. 127) reports a vulval metastasis appearing three months after a nephrectomy for a hypernephroma, and my colleague, Mr. Christie Brown, tells me he has seen the same thing. Frank also gives references to the cases of metastatic carcinoma of the vulva recorded in the literature.

Mr. CHRISTIE BROWN: Recently I saw a case showing a secondary deposit of an adeno-carcinoma of the vulva. The patient was a single woman, aged about 55, and was under the care of Mr. Gordon Luker at the London Hospital. She had had a panhysterectomy performed eighteen months previously for carcinoma of the body of the uterus, and returned with a small growth, the size of a shilling, growing from the vaginal wall, low down, just to the right of the urethral orifice. It was bright red in colour. Microscopically, it proved to be an adeno-carcinoma.

I have also seen secondary deposits of hypernephromata occurring at the vulva. The first case I saw was under the care of Mr. W. W. King, of Sheffield, in 1921. The patient came to hospital on account of a growth in the vulva about the size of a walnut. It was distinctly purple-coloured, somewhat lobulated and was growing from the anterior vaginal wall in the middle line at the vulvo-vaginal junction, i.e., just behind the urethral orifice. Microscopically, it proved to be a hypernephroma. The primary growth was found in the left kidney on laparotomy.

A second case I saw was shown to me in 1923, by Professor Croft, of Leeds. Here, again, the growth was situated in the middle line, low down in the anterior vaginal wall, just behind the urethra. It had almost an exactly similar appearance to the one I have just described. As before, this growth was proved by the microscope, and a hypernephroma was found in one kidney at the operation.

About eighteen months ago, in the out-patient department of the London Hospital, I

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saw a woman who had a growth similar in appearance and similarly situated. She complained of hæmaturia, so I at once suspected that this was a case of hypernephroma, but could detect no swelling on palpating the renal areas. However, I put her on the list to come in for treatment, but unfortunately when she was sent for I found that she had already been admitted to another hospital. I have been unable to trace her.

There is a similar specimen in the Museum of the Royal College of Surgeons. The specimen consists of a triangular flap of mucous membrane removed from the anterior vaginal wall, with a growth the size of a walnut, purple in colour, growing from the middle line at the vulvo-vaginal orifice. It is a secondary deposit of hypernephroma. In view of the situation of these growths, I have often wondered if they could have been spread by transluminal transplantation, i.e., down the urinary tract.

Standard of Results in the Treatment of Eclampsia. An Experiment in the Treatment of Eclampsia by Telephone Consultation.

By Professor W. STROGANOFF (Leningrad).

ECLAMPSIA is still one of the greatest dangers which may befall the child-bearing woman. In the United States it is responsible for more deaths than any other complication of child-bearing, if fœtal and maternal mortality are taken together. Professor De Lee [1], of the University of Chicago, states that in the United States the number of deaths among mothers only, from eclampsia, is 5,000, and from puerperal infection 6,000. As in eclampsia twice as many children die as mothers, whereas in puerperal infection the child more usually survives, the total loss of life is therefore much greater in eclampsia.

It is a sad fact that even at the present day the mortality in child-bearing is still very high. Dr. Janet Campbell [2] has published the figures for England and Wales during the four years 1919-1922. In bearing 3,279,158 live-born children, 13,465 mothers died; this represents a death-rate of 4·11 per thousand, or one in 243. These figures do not include the morbidity following child-bearing, and it is well known that many women have their health permanently damaged from this cause.

In the United States the conditions are much worse. De Lee says "25,000 women die annually here during child-birth." Mosher states that the number of labours in the United States of America is 2,500,000 a year, producing the astounding mortality of one death in every hundred. The above figures show that attempts on our part to obtain better results are urgently necessary, and that every detail in management and treatment which will produce improvement should be thoroughly investigated.

I am strongly of opinion that these terrible figures of maternal mortality from child-bearing can be reduced to a much lower level by the proper management of pregnancy and labour.

Dr. Janet Campbell states that, in England and Wales during 1922, 1,079 women died from puerperal sepsis and 556 from albuminuria and eclampsia. Dr. T. Watts Eden has stated that about 50 per cent. of the children died from the latter complication (the maternal mortality being 22·5 per cent.). These figures show that in England and Wales, albuminuria and eclampsia hold the first place among the fatal complications of child-bearing.

In Germany there is an improvement in the mortality rate from eclampsia, though many clinics still give very poor results: for instance the figures from 1907 to 1926 were as follows:—

Breuning (Tübingen)	...	...	mothers	22·7 per cent.	...	children	31·7 per cent.
Bauch (Cologne)	..	...	,,	23	,,	...	,,
Powitzer	..	...	,,	18	,,	...	
Hochenbichler	...	...	,,	20	,,	...	
Nevermann [3]	...	...	,,	32	,,	...	

In the earlier years of this period (1907 to 1926) the rate was 50 per cent., while at the same time I recorded 360 cases of eclampsia with a mortality of 6·6 per cent. of mothers and 21·6 per cent. of children. As I have just said, improvement is in progress in Germany. Zweifel [4], who until 1911 had a mortality of mothers of 15·0 to 18·5 per cent., under the Aderlass-Stroganoff conservative method of treatment, has more recently experienced a reduced maternal mortality of 8·5 per cent. in 316 cases. Leopold, who formerly had a mortality of 19 per cent. in mothers and 55 per cent. in children, with the prophylactic method has reduced it to 8 per cent. in mothers and 18·6 per cent. in children. Stoeckel [5] with rapid delivery + Stroganoff treatment, had a mortality in mothers of 8·4 per cent. in 119 cases. Waldstein [6], with the use of luminal, had a mortality of 17 per cent. of mothers and 10 per cent. of children in 117 cases of eclampsia. Nevermann, whose figures I have already quoted, states that during the period 1924 to 1926 the maternal mortality fell to between 7·5 and 12 per cent., and the mortality of children to 10 per cent.

The most remarkable results are given by Jacobs [7], though in a rather small series. Out of 48 cases of eclampsia only three mothers died, two were moribund when admitted to the clinic, and one died from inflammation of the lungs three weeks after delivery. The most remarkable thing is that not one patient in the remaining 45 cases had a single fit after the beginning of the treatment. Jacobs used the prophylactic method.

In the United States little improvement is noticed, the prophylactic treatment has been introduced only during the last ten to twelve years; for example, Professor Williams, of the Johns Hopkins Hospital, made use of a modified prophylactic method and lowered the mortality of mothers from 22·8 per cent. to 13·3 per cent. Prof. King [15] of the University of Louisiana at Tulane, using the prophylactic treatment in 25 cases of eclampsia, had only two deaths as against a previous death-rate of 34·8 per cent.; one of these was from sepsis two weeks after labour, and the other from broncho-pneumonia and nephritis.

In Japan Professor Katsuya [8] reduced his mortality figures by the use of the prophylactic method from 34 per cent. to 20 per cent. In a small series of twenty cases, treated by our method, but with luminal instead of chloral-hydrate, he had one death only.

Twenty years ago I recorded 360 cases of eclampsia [9], which I had treated with a maternal mortality of 6·6 per cent. and fœtal mortality of 21·6 per cent. I did not include the children of mothers who had suffered from post-partum eclampsia. I then said, as I had said before, after analysing the fatal cases, that the prophylactic treatment properly applied to cases of eclampsia which were neither neglected nor infected would yield a mortality of 1 to 2 per cent. only. No one believed in it at the time, but in 1913 Professor Zweifel [10], after studying ninety-four cases of eclampsia with five deaths, confirmed my statement.

On analysing cases treated by the improved prophylactic method, I now conclude that the mortality among patients that are not neglected or infected must be about *nil*. With correct treatment and diet, if infected and neglected cases are included, the number would be about 2·5 to 4 per cent. Papers by King, Waldstein, Jacobs and Hinselmann [11] confirm this opinion. An important objection to the treatment is that as yet no one has treated a sufficient number of cases (200-300);

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also, some people, fortunately only few, have had very bad results—20 per cent. mortality or over. I believe the reason for this is that the treatment was used neither strictly nor correctly. The prophylactic treatment is not fully stated in any text-book of obstetrics, even by those who consistently adopt it, as Zweifel, Lichtenstein, Seite, Williams, Fairbairn, De Lee and others. Not only is this so, but some great authorities state it incorrectly, e.g., Essen-Möller, in "Biologie und Pathologie des Weibes," and Bumm in his "Text-book of Obstetrics." It is not surprising that their results have been so bad. Most accoucheurs alter the régime of the treatment, a procedure which usually impairs the result. Many think that a fixed scheme of administration of the narcotics constitutes the essential difference between the old and the new methods, whereas my scheme provides for a variation in this routine of administration, depending on the condition of the patient. In reading descriptions of several cases in which the result was bad, I have usually found a want of individualization in the treatment. At the present time we are, therefore, obliged to confess that:—

(1) Mortality from child-bearing is still very high.

(2) One of the chief dangers both to mothers and infants is eclampsia.

(3) According to Kellogg [12] one in ten, one in five, or even one in two mothers die from eclampsia.

On the other hand, we have the statement founded on the results of 300 cases of eclampsia, and partly corroborated by the analysis of 700 cases, treated by the author himself, that mortality, including neglected and infected cases, can be reduced to $2\frac{1}{2}$ or 4 per cent., and still lower when there are no such complications. This question is of great importance, and has interested able men of all countries from Sweden (Professor Forssner) to the Argentine (Professor Zarate), and from the United States (Professors Williams and King), and Germany (Professors Hinselmann, Lichtenstein, Pankow) to Japan (Professor Katsuya). At the present time the mortality in Leningrad is very great; one woman dies from eclampsia in 1323·6 labours. All countries should collaborate in considering the question of the treatment of eclampsia. Our Academy of Science in Leningrad sets an example in this respect; it adapts both its laboratory work and its clinical researches to the everyday needs of the country.

I will now describe the way in which the mortality from eclampsia can be considerably lowered:—

There should be a sufficient number of cases of eclampsia in hospital with a low mortality. Twenty years ago I recorded 360 cases with a mortality of 6·6 per cent. During the last fourteen years I have had better results, I believe they are the greatest number mentioned in literature, namely, 300, and can be regarded as a possible standard of success at the present time. Out of 300 patients eight died (i.e. 2·6 per cent.)

In four of these fatal cases the patients were moribund when they reached hospital, and died in from three to eight hours after admission. Two died after mild eclampsia, the first had one convulsion; the other died from pneumonia and sepsis. Another patient could not be treated by the prophylactic method because of the lack of chloral-hydrate; she had, in addition, had both legs amputated as the result of an accident. In this case only out of the eight fatal cases was it impossible to control the convulsions. During the tenth and eleventh fit this patient had an apoplectic seizure and died. It is possible that her life might have been saved had it not been for the want of chloral. Post-mortem examination, by Professor Shorre, showed a heart permeated with fat, particularly on the right side, where the fat reached the trabecular muscles; generalized adiposity, bronchitis, pulmonary

hyperemia without œdema, multiple hæmorrhages in and necrosis of the liver, chronic perisplenititis, nephritis, cysts and hypoplasia of the true corpus luteum, normal post-partum uterus and recent hæmorrhage into the fourth ventricle.

To sum up, we had a general mortality of 2·6 per cent. and a reduced mortality of 0·3 per cent.

In 40·3 per cent. of the 300 cases there was not a single fit after the beginning of the treatment. In 44 per cent. there were from one to three seizures and in 15 per cent. four or more.

We observed a large number of cases of intermittent eclampsia, i.e., cases in which there was a freedom from fits for twelve hours or more before delivery. In 1916, before and during labour, we had 27·5 per cent. of these cases. Among antepartum cases there were only 63·8 per cent. of these intermittent cases. Post-partum eclampsia cases are excluded.

In five cases (1·6 per cent.) there was mental disturbance.

The mortality amongst children was relatively favourable, amounting to 16·6 per cent., but if prematurely born children, weighing under 2 kilos (about 4¼ lb.) are excluded, who died from cold, operations, etc., before treatment, the fœtal mortality from eclampsia was 6 per cent.

There was not a single maternal death due to treatment. It is known that in the treatment of eclampsia by forced delivery, approximately 4 per cent. of the mothers die as the result of the operation. There was a relatively small number of operative deliveries, namely 50 per cent. There were a few cases (1·3 per cent.) of pneumonia. Rapid recovery of the patients was general.

It seemed advisable to enlist the interest of great authorities in Vienna, Berlin and London, and I thought I should find a large enough number of eclampsia cases for a convincing demonstration, but I am sorry to say I was mistaken. Notwithstanding the interest and sympathy shown by Professors Rosthorn, Schauta and Pisacek, in Vienna, Professors Bümm, Franz, Martin, etc., in Berlin, and by Fellows of this Society in London, I obtained very few eclampsia cases in these cities. My travels, however, were not quite unsuccessful with regard to the propaganda of the method. For example, after my journey to Vienna, Leopold, Krönig, and Zweifel began to study the method; after my visit to London, Mr. Aleck Bourne used the conservative prophylactic treatment, and several American practitioners (King, Speidel) also carried it out.

The good result of my journeys is confirmed by the fact that in Germany, with the obstetricians of which country I corresponded first and most frequently, the improvement mentioned has already been manifested.

In Leningrad, as well as in Berlin and London, I used the telephone freely during the treatment of the patient; it seemed quite natural to do so with regard to cases which I had not seen. Professor Okintchitz kindly allowed me to carry on this trial in the lying-in hospital of Professor Sneguiereff, where from March, 1925, I participated in the treatment of eclampsia. The following are the results up to March 24, 1928. Out of about 11,000 admissions annually, there have been 176 cases of eclampsia, eight of which were complicated by twins. Of this number, fourteen mothers died (7·9 per cent.) and forty-two children (22·8 per cent.). In his paper Goussakoff [13] stated that the mortality of mothers used to be 16·4 per cent. and the mortality of children 31·8 per cent. Of the forty-two children who died during these three years (1925-1928) twenty-six can be excluded, thirteen died before the treatment was begun, six weighed 2 kilos (about 4¼ lb.) or under, two died from placenta prævia, and five were breech presentations. "Consequently the mortality of mothers has been lessened to 8·5 per cent. and general mortality of children to

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9 per cent. and reduced 11·7 per cent. The former mortality for these three years would have been about twenty-nine deaths in mothers instead of fourteen, the general mortality of children about fifty-eight instead of forty-two, and the reduced mortality of children thirty-four instead of sixteen."

Maternal deaths ought to be thoroughly analysed. I have analysed as follows the eight which occurred in my 300 cases:—

Two patients who died had only one eclamptic fit; each died from quite different causes. The first died from intractable hæmorrhage one hour and fifteen minutes after version and delivery.

In the second case manual removal of the placenta had resulted in rupture of the uterus; the abdomen was opened and the uterus removed. The patient died some hours later.

The third patient had two fits at home and eight in hospital, labour lasted forty-eight hours, the child presented by the vertex, the cervix being fully dilated. For some time the patient had no fits, but she was not delivered, contrary to my advice. On the fifth day after labour her temperature rose, and she died from septicæmia a month later.

The fourth patient had one fit before admission and seven fits in hospital. After delivery she had four fits: after the eighth fit venesection was performed. The fits were controlled, but the heart remained weak and the patient died after two days from double catarrhal pneumonia. By accident I had not been consulted in this case, or in the two former cases.

The fifth patient had had several fits at home in the six hours before her admission to hospital. Deeply comatose on admission, during examination under chloroform she ceased breathing. She had four fits in the first few hours after admission. The fits were controlled and the patient grew a little better under the influence of camphor and other stimulants. The life of the fœtus could not be saved as the mother's condition was so serious. She died two days later.

In the sixth case I was consulted after the sixteenth fit, when the patient was dying. As she was very anæmic, venesection could not be performed, but, as she had three very severe fits in my presence, in the last of which her breathing ceased, bleeding was carried out (150 c.c.). The fits ceased, but after a temporary improvement the patient died. It should be noticed that in this case eclampsia set in after labour.

The seventh patient had two fits at home, followed by deep coma: pulse 110, respiration, 40. She had no fits after admission to hospital. Labour did not begin. Seven hours after her arrival the patient turned blue, her breathing became more difficult, her pulse could not be felt and she died. No post-mortem examination was held, so the cause of death is uncertain.

The eighth patient had one fit at home, the second on entering hospital and eleven afterwards, two of these after labour. Her temperature during labour was 38·7° C. (101·8° F.); 40° C. (104° F.) at death. Post-mortem examination showed catarrha inflammation of the lungs and definite right-sided pleurisy.

From the statistics previously summarized it is obvious that mortality can be reduced.

I must say, however, that consultation by telephone was far from ideal in this method of treatment. There was an improvement in 1927 and 1928, when only three deaths occurred in eighty-five cases of eclampsia, namely a mortality of 3·6 per cent. (These deaths are described in cases 4, 7 and 8 above.)

I will compare this with what occurred previously in the same lying-in hospital, in and with my Standard Series. Counting in the fits of the first 176 women, published by Goussakoff (1913-15 and part of 1916), it appears that collectively they had 1,390 fits, an average of 7·9 fits per patient, whereas, during 1925-28, 176 patients had 887 fits, or five fits per patient. During 1925-1928 forty-seven cases of eclampsia, who began their fits at home, had 122 fits before admission to the lying-in hospital of Sneguireff, i.e., 2·5 fits a patient, and after admission the whole 176 had 766 or 4·3 fits a patient.

In my Standard Series, in the first 176 cases there were 731 fits, that is 4·15 per patient. Of these, fifty-eight patients had 299 fits before admission, i.e., 5·15 each. In the hospital in the whole 176 cases there were 432 fits, or 2·5 fits per patient. These numbers show that the cases of eclampsia in the hospital were more severe, but that their results were more favourable. I think this is due to the strict use of the improved prophylactic treatment by the whole staff of the institute, who had used it for thirty years, as well as to better accommodation and nursing.

As the use of this method, improved in the hospital of Professor Sneguireff in 1927-28, the results were better and the number of fits less: eighteen patients had sixty-three fits before admission (or 3·5 fits per patient), a fact which shows that they were admitted in a more serious condition, and in the hospital eighty-five patients had only 307 fits, or 3·6 fits per patients, a considerable improvement.

I must point out several details: among the 176 patients there were eight cases of twins, forty-seven patients who had fits before admission, 123 after admission and six not recorded; primiparæ 75·3, multiparæ 24·7. The fits occurred antepartum in thirty-one cases, intrapartum in ninety-three cases, post-partum in fifty cases; in two cases there is no record. Operations for delivery: seventy-six out of 124 cases; of these nine were cases of Cæsarean section, and in five of these there were complications, namely, contracted pelvis and placenta prævia. In four cases there was eclampsia only. I did not approve of the treatment in these. The other deliveries were: forceps, fifty-three occasions; extraction of breech, four; version and extraction, three; Kristeller, one; perforation of the dead fœtus, four; craniotomy, one. There were six preliminary operations: metreurysis, three, of which I do not approve; incision of cervix uteri, one; bringing down of foot, one; episiotomy, one. Complications occurred in thirty-four cases: there were thirty of suture of perineal lacerations, three cases of manual removal of placenta, and one hysterectomy for rupture of the uterus. It should be noted that in three cases of Cæsarean section there were three dead fœtuses (33·3 per cent.). In four out of the five cases of Cæsarean section the respective ages of the patients were 20, 21, 23 and 25. I am very anxious about their future labours.

In regard to a wide use of Cæsarean section in the treatment of eclampsia, the conclusion to which I have come from my observation of over 1,000 cases, as well as from the study of English, American and German literature, is that this treatment often causes death and nearly always unnecessarily injures the patient.

A few words as to the improvement of the prophylactic method: I do not look upon it as ideal. I think we can improve it as well as its results, although these are so much better than others that to many they seem unattainable. The percentage of controlled fits from the beginning of treatment was only 40 per cent. of those under my personal supervision—57 per cent., and only in Jacobs's series have they risen to 93 per cent. The last two groups are not large, and, including that of Jacobs, consisted chiefly of mild cases of eclampsia.

The use of the quartz-lamp is important in the treatment of eclampsia; its beneficial action has been described by Hochenbichler, who has advised its use in conjunction with the prophylactic treatment. It involves no danger to the heart or to the nervous system, and it produces a noticeable vaso-dilatation. Luminal has also proved useful in the treatment as a substitute for choral hydrate. Its action is similar to that of chloral hydrate, but it is less dangerous to the heart and the nervous system. Its great advantage is that it may be given by subcutaneous and intravenous injection, whereas when chloral hydrate is given per rectum there is considerable doubt as to whether it has been absorbed.

The disadvantage of luminal is its instability. The same is true of hedonal,

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which I have used in a few cases without good results, but I may have used too small doses. It also has the advantage that it can be used intravenously and has no ill effect on the heart and nervous system. Regarding chloral hydrate as a powerful narcotic, the action of which upon the vasomotor centre is the strongest, we can hardly do without it in severe cases, but after the fits have been controlled for from eight to ten hours, or in those cases in which the chloral hydrate has not been absorbed, the substitution of luminal or hedonal might be tried.

I have less faith in magnesium sulphate, which is much extolled by American practitioners. I find that its results are only mediocre—Lazard gives a mortality of 13·3 per cent. and Mayes mentions that several deaths have been caused by it.

These drugs are to be used as additions to the improved prophylactic method without affecting its main scheme of application.

Thirty years ago a method of treatment was tried which gave a maternal mortality of only 5 or 6 per cent. in cases which were nearly all infected, neglected or wrongly treated, but everyone regarded the results as accidental. Ten to thirteen years later, Leopold, Krönig, and Zweifel studied this method of treatment seriously. Already up to the present time 4,800 cases of eclampsia have been published in which the prophylactic method and its variations have been employed, and the mortality is now 10·6 per cent. This mortality is very high, although several distinguished men, namely, Strassmann [14] and Olshausen, declare that mortality from eclampsia cannot be lower than 8 per cent. This is not correct. We have ample foundation for the belief that mortality from this illness can be brought down, as a rule, to from 4 to 2·5 per cent., including neglected and infected cases.

If we take into consideration the fact that in Russia 1,500 to 2,000 women die every year from eclampsia including about 3,000 to 4,000 children; in the United States, 5,000 mothers with 7,500-10,000 children; in England, 600 women and not less than 1,200 children, we see that in these countries alone mankind loses every year about 20,000 lives. Should we not immediately resolve to find out the best treatment for this plague or shall we wait another ten to twelve years for a clearer insight into the question, during which time 100,000 more lives will be lost? I think it absolutely necessary that we should set ourselves at once to solve the question and see whether the mortality from eclampsia can be reduced to between 2·5 and 4 per cent.

That eclampsia in the United States is the most serious complication of pregnant women is quite obvious from Kellog's words [12]: "At present toxæmia is the obstetrician's unconquerable enemy, in spite of optimism. The maternal mortality in Massachusetts in cases treated in well-equipped hospitals by reputable obstetricians was 50 per cent." It is very interesting that in the United States both Kellog and Lazard think it necessary to establish a committee composed of scientists and clinicians to inquire into the question of the treatment of eclampsia, a matter for which I agitated both in Leningrad and in London some years ago.

Conclusions

(1) Mortality from eclampsia is still very great, in several countries it is frightful.

(2) During the last twelve to fifteen years, in some hospitals mortality has been reduced to between 8 and 12 per cent. by the wider application of the principles of the prophylactic treatment.

(3) The mortality still remains very high, but there is a great probability of its being brought down to one half or one quarter.

(4) The best results have been achieved by the prophylactic treatment.

(5) Hardly anyone has ever used this method, strictly or fully, or the results would have been better.

(6) The improved prophylactic treatment is rendered more difficult by the different narcotics used and many complicated details.

(7) The technique of its use is of great importance and is best learned at the bedside of the patient.

(8) If presence at the patient's beside is impossible, the telephone should be used : it is most helpful.

(9) The prophylactic treatment has assisted in reducing the maternal mortality and has reduced the fœtal mortality 8·5 per cent., even when not used very exactly.

(10) Wide and collective trial, with strict application of the improved prophylactic method, is a pressing need to-day.

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Discussion.—Major P. FLEMING GOW, I.M.S., said he welcomed any contribution to the treatment of eclampsia, which was still attended by a high mortality in Bengal, where its incidence seemed to be much greater than at home, largely owing to the ignorance of the population and their failure to appreciate the ante-natal care, which was now more widely available in the larger towns of India. In Calcutta our mortality figures had improved slightly, with the adoption of Stroganoff's treatment, but they did not even approximate to the low figures just quoted, nor was the failure to achieve a similar result due to the modification of his treatment deplored by Stroganoff, but rather to the high percentage of severe cases in which the patients had many fits and were comatose on admission to hospital. It was not their practice to do Cæsarean section for eclampsia alone, but only when there was some other definite complication and indication, such as "the undeveloped pelvis," in their very youthful primiparæ, and then the results were good only when the operation was performed early, i.e., not when the patient was exhausted by many fits. He confessed to not having previously heard of luminal being used in the place of chloral and regretted that Professor Stroganoff had not indicated the dose he had found useful. As most of their patients were comatose on admission, and required chloral in rectal saline, the amount absorbed could not be so accurately controlled as luminal given by subcutaneous injection, and therefore he would give this modification a trial.

Dr. LOUISE MCILROY suggested that a standardized treatment of eclampsia should be drawn up and published. Patients in most hospitals frequently came in as emergency cases and were treated by the junior staff and many methods were employed. Professor Stroganoff's paper was full of statistics but did not outline his more recent methods of preventive treatment. A note of these methods should be appended. Professor Stroganoff did not give Great Britain sufficient credit for ante-natal preventive treatment. When he came to London four years ago in order to have the opportunity of demonstrating his treatment, he could not find more than one or two cases of eclampsia. He made the statement that in London the prophylaxis of eclampsia was an example to be followed by other countries.

There must be a certain degree of mortality from those fulminating cases which gave little or no warning of the onset of eclampsia, and in which every method of treatment failed.

Mr. L. C. RIVETT said that he supported Professor McIlroy's plea for a standard treatment of eclampsia, as he was convinced that modification of the original Stroganoff treatment was

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responsible for the poor results. At Queen Charlotte's Hospital the original treatment had been modified and the results had not been good. At Queen Mary's Hospital for the East End the original modified treatment was kept written up in the labour department, and was rigidly adhered to. A series of forty-four cases had been admitted with only three deaths.

[In reply to a question by Mr. A. L. Walker, Mr. Rivett stated that he employed chloroform anæsthesia.]

Section of Otology.

President—Mr. J. S. Fraser, F.R.C.S.Ed.

[*May* 5, 1928.]

The Histopathology of Mastoiditis.

By J. P. Stewart, M.D., F.R.C.S.Ed.

Abstract.—Mastoiditis, a broad term, with no strict anatomical basis, includes not only an inflammation of the pneumatic cells in the mastoid bone proper, but also all extensions into neighbouring bones (zygoma, occipitalis, etc.).

Thus the anatomical distribution of the pneumatic cells plays an important part in the course of the disease. This point is illustrated by Brock's case, in which a fatal meningitis ensued from a pneumatic cell in the wall of the internal auditory meatus. According to this author's investigations on pneumatized temporal bones of all ages, 11 per cent. had such an extension normally to this site. Even if diagnosed, the impossibility of opening up such a cell is evident.

In the present paper the various stages of acute mastoiditis are described and illustrated:—

The initial change in the directly infected zone is a local rise of blood-pressure, causing dilatation of the vessels in the Haversian systems and hyperæmia of the muco-endosteum. In the Haversian systems the rigid bony walls allow of no physiological expansion, and so, as early as the third day, osteoclasts are found actively eroding the bony walls. Meanwhile the vessels in the muco-endosteum endeavour to lessen their congestion by transuding fluid into the supporting endosteum, which consequently becomes swollen and œdematous.

The next stage is characterized by a more advanced degree of osteoclasis in the Haversian systems. The muco-endosteum is infiltrated by round mononucleated cells, the change being most marked under the epithelium; the infiltrating cells push forward the epithelium and escape into the cell lumen through the rents.

At the same time softening or halisteresis occurs in the bone and a few perforating vessels are seen.

In influenzal, but not in other types, hæmorrhages into the muco-endosteum occur.

The third stage is the period of active rarefaction of the bony wall of the pneumatic space by osteoclasts and perforating vessels. This takes place because of the new pressure conditions; in the former stages there has been no real increase of pressure in the cell space, but at this stage the epithelium has largely disappeared, and the pneumatic cell has become a cavity lined by granulations and full of unorganized exudate centrally.

The next change consists in the regeneration of the destroyed tissue by new bone formation. For this a reduction of the existing pressure is required, and may be explained by an equilibrium of pressure in the vascular system—a response by the organism to new conditions. The extra space obtained by the destruction of bone is also a factor.

The whole inflammatory condition is subject to phase-change, which can turn it from one of exudation, accompanied by increase of intravascular pressure, into a more proliferative one (Krainz). The latter phase is introduced by a transudation of œdema and tissue fluid back into the veins (Korner). This process is essential for the decrease of the local

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pressure, since the only outflow of the tissue fluid from the bones is by the veins (Recklinghausen). Thus is explained the occurrence of the proliferative process in those cases in which no eruption through the covering occurs.

In the regenerative stage the remnants of epithelium subserve the function of preserving portions of the original pneumatic cell lumen.

It thus becomes apparent, first, that a certain number of the pneumatic cells must be converted into spongy spaces and, secondly, that the process will be most pronounced in a very cellular mastoid, because such a mastoid contains outlying cells in which pus stagnates and in which organization will eventually take place. There is a distinct potentiality for the mastoid process to become converted into spongy bone. Granulations grow from one cell to another, from the actively diseased zone to the less diseased parts, until they are held up by a growth of epithelium barring their further progress. When it is remembered how quickly the "proud flesh" of some mastoid wounds sometimes grows, despite bluestone applications or instrumental removal, it will be readily realised how a similar condition inside the mastoid will eventually result in the bone being converted into the spongy type. It is not maintained that this change is a common occurrence; it may be exceedingly rare but such a possibility must be stressed.

What is removed at operation depends upon the time at which the operation is carried out. If this should be late, new-formed bone, organized tissue and diseased tissue are taken away, and macroscopally no differentiation can be made between them; hence a complete clearance is necessary.

The infecting micro-organism has some effect on the course of the disease, the most dangerous being *Streptococcus hæmolyticus*.

The disease is not influenced directly by age, provided that the patient is healthy, but its course is dependent on the acuteness and severity of the infection.

Briefly, then, the first stage of the disease consists in a destructive process beginning in the Haversian canals, and then involving the pneumatic cells. The disease extends from the interior to the exterior, and the same order of progress is observed in the subsequent regenerative processes.

THE theme of this paper is by no means new, the first comprehensive article on the subject having been published by Scheibe in 1903. His opinion was unchallenged for over twenty years, until Krainz wrote his monograph on "Mastoiditis"; this author presented the subject from an entirely different aspect, and since then numerous papers have been published upon it.

Up to the "nineties" all forms of mastoid disease were included in the term "caries," as comprehensive and as convenient a term as "influenza" to-day. Politzer divided the cases into "acute" and "chronic." Bezold subdivided the acute forms into "genuine" and "those complicating a general disease," (secondary). Scheibe, on the foundations of his researches, renamed the conditions forming the latter subdivision, "genuine empyema" and "empyema complicating general diseases," empyema in his mind, being the nucleus of the disease. By "empyema" Scheibe understands an accumulation of pus under pressure originating from one or more eccentrically dilated pneumatic cells, i.e., from a preformed cavity lined by epithelium. Max Mayer substitutes "bone abscess" for "empyema," as he maintains that the purulent exudate accumulates in a cavity formed by the disintegration of tissue and, therefore, not preformed.

Since then great progress has been made in the pathology of diseases of the mastoid process. Pneumatization has been investigated by Cheatle, Wittmaack and Mouret. A study of the structure of the lining of the mastoid cells was embodied in a paper by Krainz. The effect of the closure of the Eustachian tube and its consequences to the mastoid process, were related by Scheibe and elaborated by Krainz.

The result of work directed towards elucidating the pathogenicity of the various micro-organisms met with in mastoiditis has been disappointing, and the problem has yet to be solved.

Lately, Krainz has maintained that mastoiditis is the only correct designation of this disease, since the pathological changes originate in the bone (Haversian canals) and spread secondarily to the muco-endosteum. Scheibe refutes this contention on the ground that the

disease originates in the mucous membrane of the pneumatic cell before affecting the bone and is primarily of the nature of an "empyema"; the term "mastoiditis" was misleading. Considering the nomenclature from the operative standpoint, Schlitter prefers the term "empyema" or "bone abscess." The "throbbing symptom" due to the retention of pus under pressure is one on which he places great reliance for diagnostic purposes.

In the Edinburgh Clinic the present disease has always been spoken of as "mastoiditis," and we shall continue so to designate it.

In this paper the clinical, bacteriological and histological conditions in fifty cases are reviewed; of these thirty were "genuine" and twenty "secondary." As no distinctive histological differences have been found between the two groups, all the cases have been considered together.

Of the "secondary" cases thirteen occurred after and two during influenza, one after pneumonia, one in the course of subacute nephritis, and three during scarlet fever. The "genuine" ones complicated a local lesion of the nose or throat.

The material was obtained at the time of operation from bone chips of diseased tissue; hence it represents the state of the tissues removed at the Schwartze operation. On removal the material was placed in fixative (Pick, Jores, Zenker, or picric acid), and later in Perreny's decalcifying fluid. When decalcification was complete, some of the tissue was used for frozen sections and the remainder was embedded in paraffin. Sections were stained by hæmatoxylin and eosin, by the methods of van Gieson and Schmorl, and by Heidenhain's "azan" modification of Mallory's stain.

At the time of operation a specimen of pus was taken from the mastoid cells and submitted to bacteriological examination. Considerable effort was made to demonstrate the micro-organisms in sections of the diseased bone, but, except in one case, the attempt failed. The decalcifying fluid seems to destroy the staining properties of the micro-organisms.

The subject will be considered from the following standpoints:—

(I) *Anatomical.*—Development of the mastoid process. Pneumatization of the mastoid process. Arrested pneumatization. The lining of the pneumatic cell.

(II) *Pathological.*—Changes in the bone-marrow. Œdema *ex vacuo*. Changes in the muco-endosteum. Bone removal. Necrosis. Fistulæ. New bone formation.

(III) *Clinical.*—Bacteriology. Incidence of mastoiditis. Clinical manifestations. Mastoiditis in scarlet fever. Mastoiditis in influenza.

Development of the Mastoid Process.

The mastoid process is developed from two bones; the anterior part is derived from the squamous, and the posterior part from the petrous, portion of the temporal bone. Between these two bones there is the mastoid fissure, which forms the site of predilection of a fistula. The lateral wall of the mastoid bone often contains, in its early stages, islets of cartilage which facilitate fistulous formations (Keith).

The squamous portion develops directly from membrane to bone, the petrous part from membrane to cartilage, and thence to bone. Absence of the tympanic antrum has been described (Mouret, Ruttin), but is rare. The mastoid process only takes shape from the beginning of the second year onwards. It develops in response to static conditions associated with the neck muscles, to which it gives attachment. The period of its greatest growth is at puberty.

Pneumatization of the Mastoid Process.

Eckert-Möbius divided the development of the mastoid process into three periods.

The first is the development of the lumen of the recessus and antrum, which takes place during the first year of life.

The second period is the development of the pneumatic cells, which normally takes place during the second and third year of life, at latest the end of the fourth year. If, after the fifth year, an incompletely pneumatized process is present, disturbance of the pneumatization has occurred. Alexander, of Vienna, and Steinger come to the same conclusions, although Alexander states that in exceptional cases normal pneumatization may take place up to the sixth year. Incidentally, he is strongly of the opinion that, as the Eustachian tube

is responsible for the ventilation of the middle-ear cleft, and therefore plays a very important rôle in the pneumatization of the mastoid process, adenoids, if present, should be removed before this age.

The process of pneumatization is as follows : Those marrow spaces which open towards the antrum are pressed upon by the ingrowing epithelial sac, thus producing a collateral œdema whereby the parenchymatous element of the bone-marrow atrophies, leaving only its framework of endosteal tissue. This becomes myxomatous, and is later transformed into loose fibrous tissue which then becomes pressed in a very thin layer on the osseous trabeculæ. It is surmounted by a single layer of flattened epithelium. Simultaneously, widening of the bony walls of the pneumatic cell takes place either by osteoclasts or by perforating vessels (fig 1).

The third period of development is the further distribution of the pneumatic cells in later life. The pneumatic cells diverge into the marginal zones and neighbouring compact bone, e.g., labyrinthine capsule ; the process takes place by the development of small, marginal medullary spaces in the immediate neighbourhood of a blood-vessel. In the air cells contiguous to medullary bone spaces (occiput, squamous portion of temporal bone and zygomatic process, etc.) the pneumatization is the same as described under the second period.

In a perfectly pneumatized mastoid process the cells are regular and laid down in definite arrangement ; thus, we can distinguish the various cell groups which are constant. The following anatomical grouping of the cells is according to Neumann :—

(1) Zygomatic cells. (2) Cells bordering the floor of the middle fossa. (3) Angle or petrosal cells. (4) Marginal cells, lying posterior to the sigmoid sinus plate. (5) Cells overlying the sinus plate. (6) Retrofacial cells, including those cells lying behind the horizontal semicircular canal. (7) Tip cells. (8) Very rarely, cells in the bony part of the Eustachian tube, peritubal cells.

The cortex of a well-pneumatized mastoid process is of thin compact bone, while the blood-spaces in the bone throughout it are few.

There are comparatively few completely pneumatized mastoid processes (Ruttin gives about 25 per cent.), and all types may be met with, from the completely pneumatized to the acellular. Josef Beck found in his series of cases that 58 per cent. of the mastoid processes were incompletely pneumatized. Cheatle states that 15 per cent. of mastoid processes are of the "acellular type," and 2 per cent. of the "dense infantile type."

Arrested Pneumatization.

The normal development of the mastoid air cells may be interfered with at any stage by the presence and effects of local inflammation. Wittmaack, in his extensive researches, gives valuable information on this point. In the presence of disease new connective tissue is formed beneath the epithelial layer of the advancing sac. Consequently, adhesions are formed and growth of new bone takes place, encapsulating the antrum from the spongy mastoid ; thus, by a mechanical process, further pneumatization is prevented. The adhesions form loculi in which the inflammatory process may continue, although in a quiescent form, while the affected mucous membrane may be left in a state of chronic phlegmasia ; staphylococcal infection seems to favour the chronicity and also the slow course of the condition.

Cheatle's views on pneumatization are briefly as follows. He differentiates two main types, infantile or acellular, and cellular. The infantile group is subdivided into the "diploetic infantile type" and the "dense infantile type," according to the consistence of the mastoid bone. In the infantile group the outer antral wall consists of an external layer of compact bone and an internal layer of cells. The mastoid mass is diploetic and separated from the cavity of the antrum by a thin layer of dense bone which forms the antral walls. As growth progresses, the outer dense wall and the walls separating the cavity of the antrum from the diploetic mastoid mass become thicker, so that cellular development never takes place.

This process would be, according to Wittmaack, the results of a previous otitis media, and what Cheatle interprets as "developmental" is really pathological new bone formation.

Mouret asserts that a dense mastoid process has not become acellular from the result of a suppuration which might be supposed to conduce to a progressive sclerosis of the bone and gradual disappearance of the pneumatic cells, but that pneumatic and non-pneumatic

processes are natural, anatomical forms, which rather order the course of the suppuration and are not in themselves caused by it.

Of the relationship to chronicity of the first two types, all are agreed.

It is possible that, after repeated attacks of mastoiditis, not severe enough to be operated upon, a completely pneumatized mastoid process could be converted again into a partly or wholly spongy bone.

The Lining of the Pneumatic Cell.

The recent researches of Krainz leave no doubt as to the structure of the lining membrane of the air cells.

During fœtal life a double row of ciliated columnar epithelial cells buds from the naso-pharyngeal mucous membrane, and by active growth, aided no doubt by intra-uterine pressure, hollows out and lines the Eustachian tube. At birth, when breathing begins, pressure is produced by the act of swallowing, which forces air into the Eustachian tube; this in turn forces the spreading epithelium into the middle ear and its spaces. As it grows in more

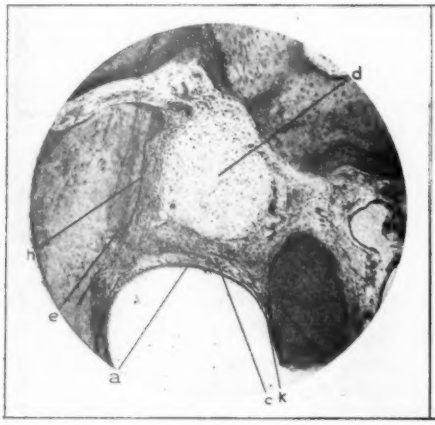

Fig. 1.

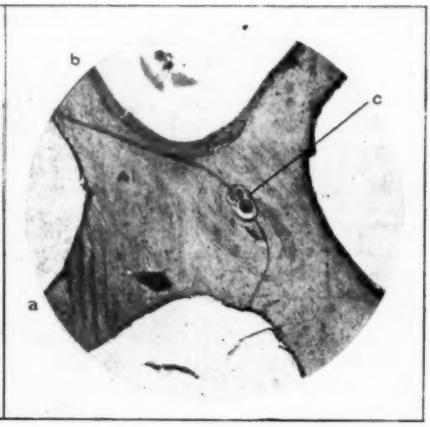

Fig. 2.

Fig. 1. *Formation of a pneumatic cell* × 40.—Pressing epithelial sac which is compressing the marrow, transforming it into fibrous tissue (*c*) at the periphery and at the centre into myxomatous tissue (*d*). The bone trabeculæ are being remoulded by perforating vessels (*e*) and osteoclasts (*h*). The epithelium of the sac is active and cubical, and of a single layer. The retrogressive metamorphosis of the subepithelial tissue into a thin muco-endosteum (*k*).

Fig. 2. *Normal pneumatic cell* × 90.—(*a*) Normal atrophic muco-endosteum; (*b*) slightly fibrous muco-endosteum; (*c*) Haversian canal.

deeply, the epithelium tends to lose its ciliated character, so that finally only the Eustachian tube and anterior part of the tympanum are lined by it; the epithelium of the mastoid air cells, on the other hand, is flattened, structurally resembling endothelium, the intermediate parts being lined by epithelium of the various types transitional between the two extremes.

This characteristic of the epithelium is analogous to that of the main respiratory tract; in the trachea it is ciliated, and in the air cells or alveoli it is flattened and has often been likened to a serous membrane.

Thus the structural lining of the mastoid air cells consists of an external, single layer of flattened epithelium with its basement membrane lying directly on the substratum of residual endosteal tissue, devoid of sensory nerves and glands, and closely applied to the underlying bone (fig. 2). It is yellow in colour from the underlying bone, but during acute disease it becomes red through the dilation of the endosteal vessels.

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Changes in the Bone Marrow.

The marrow of the mastoid process is red; it differs from the yellow in having fewer fat cells and more hæmopoietic elements.

When the marrow in the mastoid process is the site of inflammation, the reticulum swells up into a semi-transparent substance in which are a few delicate fibrous tissue cells, with long, thin processes and faintly staining nuclei. The change starts at the periphery of the space and progresses centrally. The blood-forming cells disappear, the fat cells atrophy and degenerate into a homogeneous mass. This mass is then replaced by protoplasmic cells possessing tufts of fine fibrillar processes; later, the cells become spindle-shaped and form connective tissue. This process is typical of reversion to myxomatous elements.

The next stage consists in a transformation of the myxomatous into fibrous tissue; it begins at the periphery and spreads centrally, following in the primary myxomatous change.

This fibrosis of the marrow results from a passive circulation in the presence of an irritant (Cone).

The third stage in the metamorphosis of the marrow is new bone formation. But this does not occur to anything like the same extent as in the pneumatic cells. This supports Sharpey-Schafer's statement that in regeneration of bone in the adult, participation of the marrow is much less than that of the bone itself.

The transformation of marrow spaces into spongy bone having been defined, we will now consider the reverse process by which new marrow spaces are developed.

The trabeculæ of bone arch over, meet and fuse, forming a space lined by osteoblasts and containing fibrous tissue and vessels. The fibrous tissue changes into one myxomatous in structure and from which are developed a reticulum and fat cells. With the development of these new elements, hæmopoietic cells derived from blood-vessels appear in their meshes. Marrow formed in this way, however, always remains of the fibrous type.

In none of the present series of cases has pus been found in the marrow spaces (cf., primary acute osteomyelitis). This agrees with the experience of other observers.

The changes observed in the marrow are the following: (1) Marrow tissue; (2) myxomatous-like tissue; (3) fibrous tissue; (4) new bone; and (1) fibrous tissue; (2) myxomatous tissue; (3) marrow tissue.

The marrow space can be transformed either into a spongious space or into one containing fibroid marrow.

Œdema ex Vacuo.

In order to understand fully the various processes involved in mastoiditis, a brief review of the process of œdema *ex vacuo*, though a subordinate one, is essential.

Air plays a very important rôle in the absorption of exudate from the middle-ear spaces, and its exclusion and absorption render the individual more liable to disease. This is well seen in children, in whom the air content of the middle-ear cleft is very small, and in the incompletely pneumatized mastoid in which, on exclusion, air is more quickly resorbed; on the other hand, a well pneumatized mastoid process containing more air than the antrum and middle ear becomes infected less frequently, but when infection occurs it is more dangerous, owing to the comparatively few blood-vessels.

When the blockage of the Eustachian tube or of a group of cells occurs, an œdema *ex vacuo* results. The muco-endosteum, which normally is pressed flat against the bony wall of the cell, expands to many times its original thickness; the endosteum is spread out into a looser or thicker network, according to whether it is healthy or has been the seat of previous infection; owing to the decreased tension of the supporting tissues, the vessels dilate and extravasation of plasma occurs. Where the supporting tissue of the blood-vessels consists of a large amount of well-formed fibrous tissue, the œdema will not be so intense, as the tension will be better maintained; on the other hand, pressure effects will more quickly supervene.

The patency of the Eustachian tube to the passage of air has a bearing therefore on the course of the disease, in ventilating the cavities and so favouring re-absorption.

Changes in the Muco-Endosteum.

Under the stimulus of an irritant, hyperæmia and resulting transudation of fluid into the soft tissues are produced, whereby the lining of the pneumatic cell becomes swollen and thickened. According to Scheibe, the swelling may be so great that the muco-endosteum

becomes eighty times its original thickness. This swelling is due principally to back pressure and consequent transudation from the vessels in the neighbouring Haversian systems, and the œdema *ex vacuo* is only of minor degree.

Thus, as in other tissues of the body, the initial stage of mastoiditis is an increased blood-pressure. The Haversian canals are rigid, bony tubes and the vessels in them feel the effects of the increased pressure first. A certain degree of liberation of pressure can be obtained through the medium of the lining of the pneumatic cell, and up to a point this acts as a safety valve; but pressure soon manifests itself by the presence of osteoclasts. Even three days after the onset of the inflammatory process, the effects of pressure may be plainly seen.

Although the fibrous tissue in the endosteum is small in amount, it is not dense and can therefore contain considerable œdema in its meshes. But in the incompletely pneumatized mastoid, although the fibrous tissue is more abundant, it is denser in structure and cannot expand to contain the same amount of fluid. On the other hand, the blood-supply of the perfectly pneumatized mastoid is scanty, while in the partially pneumatized mastoid it is good. This more than balances the lesser capacity of the latter type for œdema, since the raised blood-pressure can be more readily stabilized and the infection more readily combated, as evidenced by the greater cellular infiltration. The single layer of flattened epithelium becomes cubical and swollen without any corresponding increase in the size of its nucleus. The epithelium may assume a mammiform or wavy outline, due to being pushed outwards by the accumulation of infiltrating cells beneath it. These cells make their way into the lumen of the air cavity, either by passing through the interstices or by erupting through the epithelium.

The capillaries in the muco-endosteum become engorged and, by active budding, new ones are formed. Polymorphonuclear leucocytes make their way through the vessel walls, giving a perivascular infiltration; meanwhile, a transudation of fluid from the blood into the tissues takes place. The lumen of the air cell contains at first a serous and later a purulent cellular exudate. The inflamed muco-endosteal surfaces may meet, thus occluding the osteum of the cell or series of cells; the consequent increase of pressure may be shown by the presence of osteoclastic activity or other phenomena. This constitutes the empyema of Scheibe, "the danger zone," and is a condition seen mostly in the second and third week of the disease. Air cells containing pus not under pressure, Scheibe designates "simple cell suppuration." This condition may occur, for example, in the peripheral smaller mastoid cells.

All the air cells are not affected to the same degree. One cell may be acutely inflamed, while the adjoining cell is only slightly affected. The zone of reaction occupies a greater radius than that of the actual inflammation. Thus an area of advanced destruction is surrounded by one in which constructive processes are occurring. In almost every case examined, new bone, or at least active osteoblasts have been demonstrated, and as the latter predominate, mastoiditis has come to be considered a sclerosing disease.

The conditions necessary for commencing organization obtain when the exudate becomes "stagnant," less fluid, and fibrinized. At many points, through gaps in the epithelium, young blood-vessels sprout from the muco-endosteum and, together with elongated fibroblasts, form tissue bridges. The formation of granulation tissue thus proceeds from all sides (fig. 8). As organization progresses, the zone of granulation advances further into the exudate and the fibroblasts become shorter and thicker as they are converted into connective tissue (fig. 10). The stage of organization at any place can be made out by observing the degree of differentiation reached by the fibroblasts.

The epithelium is stimulated to proliferate by the process of organization, and grows along the tissue bridges, its chief aim being to conserve as much as possible of the original lumen of the air cell. These bridges may connect together by lateral shoots and thus enclose a space which is rapidly clothed by epithelium. These spaces lined by epithelium are termed pseudocysts (Döderlein), or, from their resemblance to a string of pearls, designated "pearls." If these "pearls" are in direct communication with spaces containing air, they remain and so conserve a portion of the original lumen. Further, if the "pearl" contains exudate and this is not absorbed, it remains as a true cyst. Lastly, if an attempt is made to organize the exudate in the pearl, it becomes obliterated in the process.

In the process of organization of the exudate, new tissue is formed and the total mass increases in bulk, but when fibrosis is complete shrinkage takes place (cf., scar tissue). In

each case pressure is exerted on the "pearls"; these may become flattened and appear as "epithelial ropes," or the epithelium may degenerate and the space be filled completely by fibrous tissue (fig. 10). It should be noted that before the pneumatic space can be completely obliterated as such, the epithelium must disappear, and this can quite easily take place in the manner outlined above.

Once the epithelium is destroyed, the power of resorption is lost. In regard to chronicity, this is of much greater importance when occurring in the middle ear than in the mastoid air cells.

It must not be forgotten that this process of organization is one of proliferation of endosteal tissue, that is, of potential bone-forming tissue; new bone formation, therefore, is one of the most striking structural features of the disease (fig. 4).

When, owing to the reduction of pressure, the conditions become favourable, new bone is formed. This is principally reticular and in many cases reaches almost to the epithelium, if remaining. Therefore, one of the possible end-results of mastoiditis is a spongy bone transformation.

Bone Removal.

There are three methods by which bone may be removed, namely, by (1) halisteresis, (2) osteoclasts, and (3) perforating blood-vessels.

(1) *Halisteresis* signifies a decalcification of bone through the agency of the bone fluids.

Scheibe found that the spongy transformation of bone took place only in the fundamental and interstitial—not in the Haversian—lamellæ; further, that the spongy converted bone always formed a sharp line of demarcation at its junction with normal bone; this is well illustrated in fig. 5.

On investigation, several of the sections, especially those from cases of short duration, show a want of sharp definition of the edges of the bone. This is not uniform, however; in some places the lack of definition merges into and even beyond the cement line. This shows that the process is one of absorption, since, if it were one of deposition, the new bone would be in front of the cement line and added to it.

Fibrillation of the bone margins with no cells to account for it supports halisteresis. Further, many of the bone cells become vesicular and surrounded by a clear zone, with loss of their canalicular processes, a picture strongly suggestive of halisteresis. (Compare fig. 6 with fig. 7.) Their vitality becomes affected, and consequently the bone under their immediate control loses vitality and becomes more vulnerable to the attacks of the resorbing processes.

Halisteresis takes place early and is confined to the first ten days of the disease. The absorption of mineral salts softens the bone; its tissue components are then removed by osteoclasts or by perforated blood-vessels (see later).

Halisteresis is present in 14 per cent. of the cases in the series.

(2) *The removal of bone by osteoclasts* is so common a phenomenon that a brief description will suffice.

Osteoclasts are not specific formations, but are commonly formed by the fusion of endothelial cells. The stages of their formation, from the budding of the cell from the wall of the capillary to the fully-formed giant cell in its scalloped-out space—Howship's lacuna—may be followed in fig. 8.

The activity of the osteoclast is not to be measured by the number of its nuclei, but by the surface relation which these bear to the total volume of the cytoplasm. When the osteoclast shows double-outlined fine borders and disappearance of its nucleus, it is waning in activity and on the verge of disintegration. The structure of the osteoclast and its mode of action is well brought out in Fig. 8. The syncytial cytoplasm of the cells has a striated border which is fused to the bone. In its close approximation to the bone, this border may be transformed into fine cilia-like processes which interdigitate with the minute bone fibrils (Dawson).

The action of the osteoclast appears to be biochemical. According to Nicholson, "Oxidation in such a tissue is rapid and a large amount of carbon dioxide is given off by it." Carbon dioxide is, according to Hofmeister, the great solvent of lime salts within the body. The solution of bone is therefore rapidly effected in the immediate vicinity of the osteoclasts.

The functions of the osteoclast are said to persist longer in secondary cases and are active under a pressure in which apposition bone is formed in a more resistive organism.

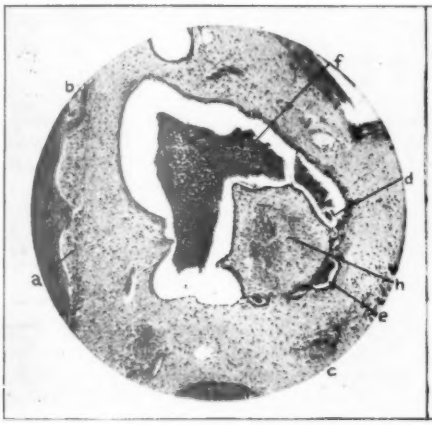

FIG. 3.

FIG. 4.

FIG. 3. *Plastic changes in air-cell lining* × 90.—(*a*) Erosion of bony wall; (*b*) commencing reticular bone formation; (*c*) proliferated fibrous endosteum; (*d*) tissue bridges growing into exudate; (*e*) "pearls"; (*f*) exudate; (*h*) organized exudate.

FIG. 4. *Bone formation in pneumatic cell* × 90 (*a*) Concentration of fibrous tissue with osteoblasts in its meshes to form reticular bone; (*b*) lumen of pneumatic cell; (*c*) osteoblasts surrounding growing margin of new-formed bone; (*d*) future Haversian canal; (*e*) osteoblasts laying down lamellar bone on (*f*) cement line.

FIG. 5.

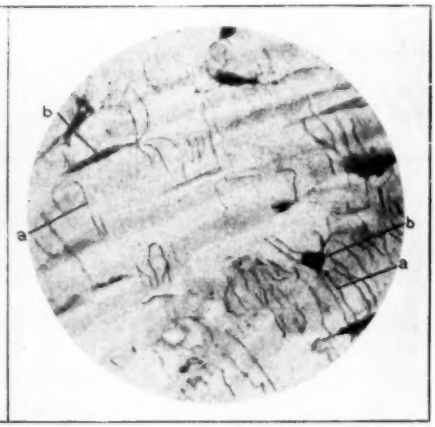

FIG. 6.

FIG. 5. *Bone removal by halisteresis* × 90.—Shows Sharpey's fibres with solution of the ground substance. (*a*) Sharpey's fibres; (*b*) Haversian canal; (*c*) medullary space; (*d*) proliferated endosteal cells and osteoblasts. Note the sharp definition between the altered softened bone and the healthy bone. Haversian lamellæ are unaffected.

FIG. 6. *Normal bone cells* (*b*) *with canaliculi* (*a*) × 850.—(Spindle-shaped, parallel canaliculi arising uniformly from the long axis.)

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Rarefaction of bone takes place at the end of the first week and is well advanced during the second week; it diminishes after this, although in one of the cases of the present series it was well marked three months after the onset of the disease.

Evidence of osteoclastic activity is found in 74 per cent. of our cases.

(3) The third method of bone removal is *by perforating blood-vessels* (fig. 9).

In mastoiditis the perforating vessel appears to be the dominating factor in bone resorption. Almost every section examined has given evidence either of the process actually taking place, or the structural changes have been such as to show that it had occurred. The perforating vessel acts not so much by its own powers of resorption as by exposing a new and greater area of the bone to osteoclastic activity.

Perforating vessels take an active part in the inflammatory process from almost the start of the disease, since their presence was well seen as early as the third day. Their presence, or indubitable evidence that they had been present, is seen in 90 per cent. of the cases of the series.

Necrosis.

Necrosis in genuine cases of mastoiditis is very rare; Krainz reports one case only out of a series of one hundred, and Otto Mayer's series does not provide a single example. When present, necrosis generally occurs in cases of mastoiditis complicating a systemic wasting disease such as diabetes, tuberculosis, etc., and, in such, is found in the very young or in old age.

In this series of cases no necrosis occurs.

Fistulæ.

Fistulæ form only when peripheral pneumatic cells are present, that is, when they are in close proximity to a free surface, either cortex or cranium. When the natural paths for drainage become occluded either by osteoplastic changes or by a plug of granulation tissue, the pus in a cell or collection of cells which have been converted into one common cavity cannot escape. Consequently pressure conditions are much more acute and, following the line of least resistance, an outlet to the surface is excavated and the pus escapes under the periosteum or dura mater. In the case of the former the canal is narrow, whereas in the latter it is much larger. A thick outer bony cortex predisposes the fistula to open inwardly. Fistulæ never lead from the antrum and the cells immediately adjacent. Apart from infants, where an obstruction to the aditus ad antrum may take place, there can be no retention of pus in the antrum and the cells immediately adjacent to it; this is due to free drainage into the middle ear and thence through a perforation in the tympanic membrane, if present, or by the Eustachian tube; hence fistulæ from these air cells never occur.

In infants a fistula is more liable to pass outwards, as the antrum is placed against the cortex which often contains islets of cartilage; or, secondly, the sutures may be still open.

It is usual to find only one fistula in the cortex; if two or more are present the case is probably one of the "secondary" group. Macroscopically, the fistula is often filled with granulation tissue; microscopically, its edges are deeply eroded by active osteoclasts—indicating a persistence of the pressure—and the lumen is filled with proliferating endosteum. The formation of the fistula does not seem to influence the pre-existing vascular pressure.

In the present series, a subperiosteal abscess was present in seventeen cases (seven male, ten female). Of these, fifteen occurred in genuine and the remaining two in secondary cases; fourteen took place after the third week of the disease; and in nine an intracranial infection was a complication.

New Bone Formation.

New bone may be laid down in lamellar formation (apposition bone) or in reticular formation. After infancy the first process normally takes place within the body; the second is of a primitive type and, apart from embryonic and infantile tissues, is seen only in pathological processes.

It is interesting that Robison and Soames have found evidence of a special enzyme in bone which brings about an orderly precipitation of the calcium salts of the blood within newly-formed bone matrix.

Lamellar Bone.—The osteoblasts arrange themselves in one or more rows along the original margin of the bone; there they secrete osseomucin, and becoming embedded in this

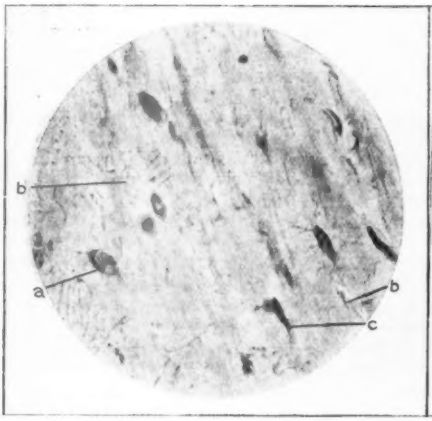

FIG. 7.

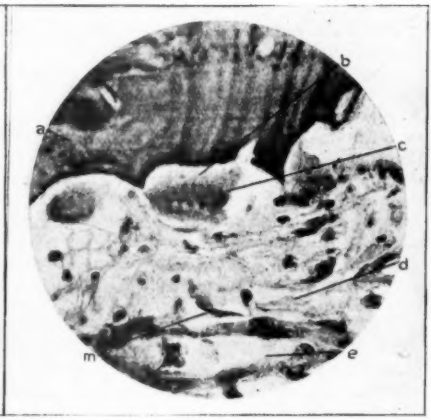

FIG. 8.

FIG. 7. *Degeneration of bone cell (b), vacuolation of its space and disappearance of its nucleus and canalicular processes* × 850.—(*a*) Similar changes not so advanced; (*c*) normal cell.

FIG. 8. *Bone removal by osteoblasts in pneumatic cell* × 350.—(*a*) Old bone; (*c*) giant cells with syncytical border (*b*) in contact with bone hollowing out Howship's lacunæ; (*d*) proliferated endosteum; (*e*) capillary. Note endothelial cells (*m*) passing towards bone to join the osteoclast.

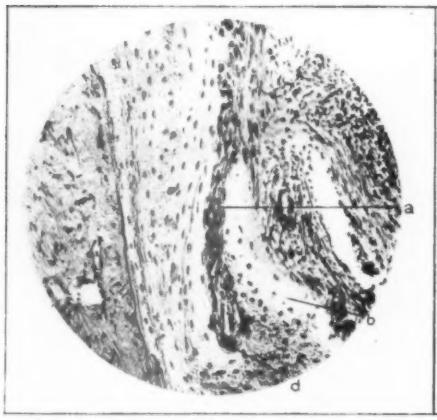

FIG. 9.

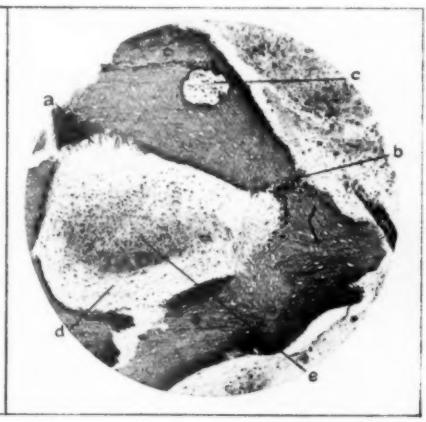

FIG. 10.

FIG. 9. *Bone removal by perforating vessels* × 90.—(*a*) Perforating vessel growing into (*b*) old bone; (*c*) proliferated endosteum; (*d*) osteoclastic surface of resorption of the capillary wall.

FIG. 10. *Pneumatic cell* × 90.—Now filled with proliferated endosteum (*d*) and organized exudate (*e*). Osteoclasts are eroding the bony walls and causing a fibrillation of the bone margins (*a*). Especially active at (*b*). Haversian canal (*c*) eroded by osteoclasts, where the bony party wall of the pneumatic cells is being resorbed.

are converted into bone corpuscles. Calcium salts are then deposited. The relation between the calcified and uncalcified portions gives one some indication of the rate of growth of the particular bone.

Reticular Bone.—As already stated, this is an embryonic type of bony formation and is analogous to bone developed in membrane.

Vascular dilatation indicates waning resorption and the first signs of new bone formation is a grouping of the osteogenic fibre bundles (fig. 4). Later, active osteoblasts are found around the fibre bundles and within their meshes, and as a result of the secretion of osseomucin the bundle becomes a homogeneous mass. At the growing points of the bony trabeculæ thus formed the fibres are continued into the general mass of fibrous tissue surrounding the area, while at the base they are continuous with the parent bone and thus acquire permanent vitality.

These bony trabeculæ may reach almost to the epithelium of the pneumatic cell (fig. 4): thus the cell, though lined by epithelium, can never return to its former dimensions. The part the trabeculæ take in the formation of secondary marrow spaces has been already considered.

By the general laws of development, imperfect forms are gradually replaced by those more perfect, so that the trabeculæ become transformed into lamellar bone.

The formation of reticular bone is a response by the organism to the great stimulus of the disease; apposition bone cannot be formed quickly enough, therefore the more quickly-growing and primitive type is produced.

In the present series of cases no evidence has been found of new bone formation within the first few days after onset of the inflammation. The osteoid edges which Mayer and Beck claim to be new bone formation are a zone of decalcification; from its arrangement, its irregularity and the inactive cells which line it, there is no doubt that the changes here are the result of halisteresis.

Great hyperæmia is induced beyond the limits of the actual infection, and this stimulates the osteoblasts to rapid proliferation. The area of bone formation is greater, therefore, than that of bone destruction. The new bone formed more than compensates for the eroded portion, and this has an important bearing on pneumatization.

The cement line is an important guide to the condition existing before new bone has been formed. By following its outline, evidence of past osteoclastic activity and of perforating vessels can be obtained. On the other hand, if the cement line is regular, with new bone set on it, the new bone is a response to an irritant and not reconstructive after erosion.

In two cases of the present series formation of new bone (one apposition, one reticular) had occurred by the seventh day of the disease; during the second and third weeks the production is progressively commoner, while during the fourth week it is most abundant.

Bacteriology.

Bezold maintains that the kind of micro-organism has no influence on the liability of the bone to be affected, or on the subsequent course of the disease, but this statement must be modified in the light of present knowledge; probably Gradenigo's dictum that the clinical symptoms are not in direct relationship to the kind of micro-organism would be more correct.

The figures for the present series are in eleven cases *S. pyogenes*, in fourteen *S. hæmolyticus*, in four pneumococcus, in one *Staphylococcus aureus*, in one *Pneumococcus mucosus*, in one *Streptococcus mucosus*, in three *S. pyogenes* and pneumococcus, in two *S. pyogenes* and *Staphylococcus aureus*, in two a Gram-negative cocco-bacillus. No micro-organisms were isolated from the remaining eleven cases, owing to the time which elapsed before the material could be examined bacteriologically.

The intracranial complications associated with these micro-organisms were: Of the eleven cases associated with *Streptococcus pyogenes*, one developed perisinus abscess; of the fourteen with *S. hæmolyticus*, four developed perisinus abscess, one extradural abscess of middle fossa, one acute purulent lepto-meningitis, one Gradenigo's syndrome, and one septicæmia; of the four with pnemococcus, one developed perisinus abscess and one died of cerebral œdema; the one with *S. mucosus* was complicated by perisinus abscess; of the three with *S. pyogenes* and pneumococcus, one had perisinus abscesses and one died from exhaustion follow-

ing pneumonia; of the two with a Gram-negative cocco-bacillus, one developed perisinus abscess; of those cases associated with *Staphylococcus aureus* (one), with *Pneumococcus mucosus* (one), and with *Staphlococcus pyogenes* and *Staphylococcus aureus* (two), no intracranial complications developed.

It is thus seen that the *Streptococcus hæmolyticus* is the most dangerous micro-organism as regards intracranial complication, and is almost always found in scarlet fever and to a lesser extent in influenza.

The pus from a case of acute mastoiditis is odourless; its consistence depends upon the infecting micro-organism, and has an important bearing, therefore, on the drainage or stagnation and subsequent organization. Thus, *Streptococcus mucosus* gives a thick, glairy pus, which is apt, therefore, to be retained and "stagnate"; further, the quiet course of the disease also favours retention of the pus. In contrast to this is the violent, profuse, pulsating discharge in an acute otitis media due to *S. pyogenes*.

In this connexion it is of interest to notice that Urbantschitsch maintains that *S. mucosus* may present morphologic variations. He indicates that the coccus may pass into transitional form or forms, recognized as definite types, e.g., *S. pyogenes*. In certain cases he has isolated the *Diplococcus mucosus* at one time, *Streptococcus hæmolyticus* at another, and finally has recovered *B. mucosus* in a third culture.

General Incidence.

The series consisted of 50 cases, 29 females and 21 males.

Ear Affected.—Right ear, 30; left ear, 20.

Age in Decades.—First, 15; second, 8; third, 7; fourth, 11; fifth, 2; sixth, 4; seventh, 3.

According to Heine, on account of the osteosclerotic character of the mastoid process in patients over 40 years of age, mastoiditis occurs with more difficulty; in sucklings, on the other hand, in consequence of the anatomical structure of the middle-ear spaces and of the delay of spontaneous perforation through the membrana tympani, extension of the inflammation from the middle ear to the mastoid process is greatly favoured.

Operation in Relation to the Period of the Disease.—In first week, 7 cases; second week, 12 cases; third week, 8 cases; fourth week, 6 cases; fifth week, 4 cases; sixth week, 5 cases; seventh week, 1 case; eighth week, 3 cases; ninth week, 2 cases; three months, 1 case; six months, 1 case.

The earliest operation took place on the third day. Intracranial complications occurred in 17 cases. Subperiosteal abscess occurred in 17 cases.

Deaths.—One from meningitis; 1 from cerebral œdema; and 2 from general exhaustion.

The actual mortality from the mastoid disease was 2 per cent.

Seasonal Incidence.—January, 1 case; February, 10 cases; March, 4 cases; April, 8 cases; May, 10 cases; June, 2 cases; July, 3 cases; August, 3 cases; September, 0 cases; October, 1 case; November, 1 case; December, 7 cases.

Clinical Manifestations.

The time at which operation was indicated was fixed by one or more of the following conditions:—

(1) When the discharge continued for more than five weeks and was pulsating and profuse. If the discharge persists for some time after an acute otitis should ordinarily have cleared up, it is coming from the mastoid cells (Bezold). In any case it is a generally recognized treatment to drain the middle ear through the mastoid.

(2) Continuous pain behind the ear; this denotes an involvement of the periosteum and occurs later than the onset of the mastoiditis, since the inflammatory process extends from within outwards. Pain may be present early, but is not intense; it is due to a slight tension in relation to the periosteum and disappears after one or two days when the existing blood-pressure becomes adjusted.

(3) Sensitiveness to pressure over certain points: (*a*) over the antrum; (*b*) over the posterior border of the mastoid; (*c*) over the posterior border of the tip of the mastoid process.

(4) Œdema and redness of the external soft parts (erection of the auricle) and subperiosteal abscess.

(5) Sagging of the posterior meatal wall.

(6) Throbbing or "beating" noises which do not clear up in two weeks.

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Scheibe explains these noises thus : They are worse at night on account of the passive hyperæmia due to the low position of the head in bed. The inflammatory phenomena occur in both the mucous membrane and the medullary tissues ; these structures communicate with each other by vessels and both of them are surrounded by bone, a tissue which is not compressible ; thus, resistance to the pulse wave is exerted, and this is manifested by a sensation of pulsation or beating.

Pus under pressure in any part of the body exhibits this "throbbing" (cf., whitlow) and it is one of the most valuable and absolute signs in the diagnosis of empyema. As these "beatings" are synchronous with the pulse, the patient's statements on this point can be easily checked.

(7) Intracranial symptoms.

(8) Continued fever : In the adult, an ordinary case of mastoiditis is unaccompanied by fever ; when fever is present, great care must be taken in the examination. In children, however, fever is not uncommon in the first few days of the illness. Alexander gives 101° F. as the limit for a temperature of an uncomplicated mastoiditis.

(9) X-ray picture : A negative plate is not reliable and only positive results are to be regarded. Positive results are obtained in only 58 per cent. of cases (Krainz).

(10) Some authorities consider that migraine occurring in a patient in whom discharge from the ear has recently developed, is an indication for operation.

It must be remembered that a normal tympanic membrane does not exclude disease of the outlying mastoid cells, which may end fatally (vide Brock's case).

The time to operate is of great importance. Naturally, it is decided on clinical grounds and by the opinion of the operator ; hence, there is not much uniformity in this matter. The figures of the present series show the greatest number (52 per cent.) were operated upon in the first three weeks, the majority of those (24 per cent.) being performed in the second week.

Mastoiditis in Scarlet Fever.

In this exanthema mastoiditis presents two types. The one is comparatively mild and really belongs to the "secondary" division of the classification at the beginning of this paper. It rarely occurs before the sixth day of fever, and is ushered in by a sharp rise of temperature ; otherwise the clinical symptoms are similar to those cases which constitute the main group.

The microscopical changes are the same as those observed in all the cases of the series under review. The prognosis and the mortality-rate also do not differ.

While the above is the commonest form in which mastoiditis occurs in scarlatina, there is an aggravated type, "mastoiditis necrotica scarlatinosa." It is distinguished by commencing about the second day of the disease and, according to Manasse, is associated invariably with tonsillitis and pharyngitis necroticans. The patient is profoundly toxæmic, and the aural conditions, which are often bilateral (potential deaf-mutism), may be masked by the general symptoms. The microscopical changes are in keeping with the severity of the condition, as indicated by the nomenclature.

Three cases obtained from scarlatina are included in the present research. They fall into the first or mild type, and the only features in which they differ from the rest of the cases of the type are in the destructive processes being more advanced and the reparative process slower.

Nager's statistics on this subject are interesting : Of 750 scarlatina patients he found 59 per cent. with normal ears and 41 per cent. with diseased ears. In only 30 per cent. of the last group was the ear condition directly due to scarlatina. Therefore, 13 per cent. of the total number of patients had purely scarlatinal ear affections.

Mastoiditis in Influenza.

As in scarlatinal cases, so in influenzal cases a distinction must be made between mastoiditis occurring in the course of the disease and mastoiditis directly due to influenzal influences. Here, again, the former cases fall into the category of those "secondary" cases and call for no further description.

The genuine influenzal cases are met with in the first days of the disease and are severe. The usual symptoms of acute mastoiditis are very intense and the case often comes to operation early.

Macroscopically, at operation the lining of the pneumatic cells is swollen, dark red or purple in colour, and a small amount of purulent secretion is present. The bone is hard and the tissues bleed readily and profusely.

Microscopically, in the endosteum of the pneumatic cells are hæmorrhages so large that they mask the structure, but the membrane is not so much thickened as in the "genuine" type.

The epithelium may be stripped off by the extravasation of blood, and again in fulminating cases it may be partially necrosed. The lumen of the air cells contains free hæmorrhage, and shortly afterwards pus cells appear.

The further progress of the condition is characteristic of a "secondary" type.

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Discussion.—Dr. J. S. FRASER (President) said that Mr. Stewart had suggested that a pneumatic mastoid might, as a result of inflammation, become a dense one. He (the President) thought this might possibly—but very rarely—occur. In a case of severe mastoiditis in the pneumatic type of bone, the inflammatory process would be almost certain to come to the surface over the antrum or to break through the tip or the posterior root of the zygoma, or at some other point and so become a manifest mastoiditis which required operation.

From this paper it appeared that there were two things to be considered in mastoiditis. First, the inflammation or empyema of the air-cell itself; secondly, the changes which occurred in the Haversian systems. The first could be seen macroscopically at operation, but to many clinical otologists the widening of the Haversian systems by osteoclastic formation might be new.

Mr. SOMERVILLE HASTINGS asked on what evidence Mr. Stewart described it as a continuous process. Had he the exact history of the mastoid symptoms in each case?

Dr. KERR LOVE said that there were cases in which mastoiditis seemed to have converted a large pneumatic mastoid into a process in which it was difficult to find the antrum at all. That might have been due, in some of the cases, to imperfect surgical technique, as in discovering a very small antrum. His belief was that the mastoid antrum was present in every case, though he had met with cases in which he could find nothing but a single cell. That was before he radiographed the mastoid process—in the bone itself, not in the patient. When he found that the mastoid process, in practically every case in which he did this, was almost entirely composed of cells, he became very doubtful with regard to this process of conversion of a pneumatic process into an almost solid mastoid process. The present demonstration seemed to show how it came about, and the question was whether it was as rare as the President said, and whether it might not be the result of disease.

Mr. C. S. RIDOUT said that some time ago he had had a patient (a colleague's son) who, while at school had suffered from pain in the ear and over the mastoid. No active treatment had been carried out, and when he returned from school the ear was still rather painful, though the temperature was not raised. There was tenderness at one spot over the mastoid, but the middle ear appeared normal. A radiogram showed some haziness at one spot, at the posterior aspect of the mastoid. He (Mr. Ridout) had advised operation. In one group of cells a small quantity of pus was found; the remainder of the mastoid was healthy. He (the speaker) thought there had been otitis media spreading into the mastoid, and that this had cleared up, leaving the posterior group of cells infected.

Mr. STEWART (in reply to Mr. Somerville Hastings) said that the results were obtained from material from fifty cases, operated on at periods varying from three days to three months, so that a good idea of the changes in the mastoid process was obtained.

In reply to Dr. Kerr Love, absence of the mastoid antrum had been but only rarely.

With regard to the density of the mastoid: if an otitis media developed in infancy, new bone processes were formed in the cells around the antrum, hindering the epithelial sac from going further, so that the mastoid bone never became hollowed out. He held otitis media responsible for the condition.

It was impossible to differentiate between diseased and healthy tissue during operation. Neumann's advice was to remove all the tissue so as to be on the safe side. Occasionally the operator could not get hold of some cells around the labyrinth. According to Bezold, with acute otitis media there was also an acute inflammation in the mastoid air cells, and therefore there could be no localization of the disease such as there was in appendicitis.

In those cases in which, on opening the mastoid, one found plum-coloured granulations and no pus, the condition was usually due to influenza, and one should wait as long as possible before operating. One school went in for early operating, the other school believed in waiting as long as possible. He inclined to the waiting policy, so that the disease process might become shut off by a healthy barrier. An exception was to be made when the condition was critical and there was danger of an intracranial complication.

A Case of Spherical Bulging of the Floor of the Third Ventricle, Secondary to Internal Hydrocephalus and Simulating a Pituitary Tumour.

By R. GRAHAM BROWN, F.C.S.A.(Brisbane).

AN error in diagnosis is no excuse for not giving details of an interesting case. V. K., a girl, aged 13 years and 9 months, was admitted to a private hospital in a very low state of health. The following history was given: The illness began with a fainting attack two years previously. As a child she had continually suffered from headaches, and in infancy was very "nervous". Previous to the present illness she was healthy, and "played about as other children did." She walked in a "funny"

way for about the last year, and had been treated by various doctors since. For the last six months she had been under a herbalist, who said she was suffering from lead poisoning. There was a year's history of vomiting, and the attacks used to occur at 4 a.m. Other attacks occurred during the day, but at no special times. For days at a time she was free from these attacks, but these intervals were never longer than a week. The patient complained before the vomiting attacks that "things went round," and during some attacks she had fallen down. Certain foods tended to bring on vomiting, whereas she could eat fruit heartily. For the last two months the attacks became worse, and in the night about one month ago she had a very bad attack, when "she stiffened out and became unconscious." "She appeared as if dead." Recently she had been losing her vision. She had not grown more rapidly than normal, but "periods" had not yet appeared. She had difficulty in emptying her bladder, but there was no history of polyuria. Her condition upon examination was as follows: Temperature 98° F., pulse 100, respiration 28; countenance dull; the lower jaws showed some enlargement, with splaying outwards of lower margins; upper jaw broad, the zygomæ and maxillæ definitely enlarged Her speech was slurred and she was languid. Her breasts were flat and showed no development; no hair in the axillæ, but some fine hair on the pubes. The sphincter face muscles were normal, and the jaws descended in the mid-line; the tongue was protruded straight, and the palate moved normally. The pupils were semi-dilated, but reacted to light and accommodation: no strabismus or nystagmus. The fields of vision, taken roughly, were very constricted, the right side more so than the left. There was no deafness, no incoördination or loss of muscle sense in the upper extremities, no dysdiadokokinesia, and no tremors. No loss of strength was apparent; the abdominal reflexes were present, and the knee-jerks appeared slightly exaggerated. There was no ankle clonus, and no plantar reflex could be elicited. Romberg's test, with the head in all positions, was negative, but on walking there was a slight tendency to fall and to deviate to the left. The heart, chest and abdomen were normal. The urine contained a trace of pus, and was of specific gravity 1018.

An ophthalmic surgeon reported as follows: "Right vision, $\frac{6}{36}$, 1 letter; left vision, $\frac{6}{24}$. Myopic astigmatism of moderate grade. Pupils active and equal. Power of convergence almost absent. Bilateral papilloedema present, but swelling not excessive, the right being worse than the left. No hæmorrhages, but considerable engorgement. The right field is very contracted and the left shows temporal hemianopsia."

The radiologist reported: "There is evidence of greatly increased intracranial pressure. Enlargement of the pituitary fossa and erosion of the posterior clinoid processes indicate a pituitary cyst as the most likely cause."

A Wassermann test was returned as "unreliable." Three days after admission, the patient, whilst being photographed in a standing-up position, gave way a little in the knees, became stiff, extended and supinated her arms and hands, bent over to the left and threw her head back. She was stiff when carried to bed, rolled over on her left side, gave a shrill cry and became unconscious. This state lasted for about thirty seconds. When she regained consciousness she lay on her back, looked pale and gave all the appearance of suffering severe pain in the head. This performance was repeated later when she was again placed upright. Owing to this it was decided not to attempt the labyrinthine tests. A provisional diagnosis of "cerebellar tumour" was altered to "pituitary tumour." As the patient appeared to be going downhill, delay was not thought advisable and three days later, under local analgesia (preliminary "allonal" and morphine sulphate gr. $\frac{1}{12}$ with atropine sulphate gr. $\frac{1}{150}$, and packing the nose with 8 per cent. cocaine and $\frac{1}{5000}$ adrenalin

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gauze and subsequently injecting 1 per cent. solution of néocaine (Corbière) through the spheno-palatine foramina) a submucous resection of the nasal septum, with middle turbinectomies, was performed and the anterior wall of the pituitary fossa opened over an area of about the size of a finger-nail. The dura mater of the fossa bulged through the opening and a needle was easily inserted. Six c.c. of clear fluid dropped from the needle and there was a further loss of about 2 c.c. (The technique of Hirsch was followed.) Cerebro-spinal fluid was then suspected and the operation was stopped. Examination showed the fluid to contain globulins and albumins, but no sugar. Twenty-four hours after operation, the patient complained of headache and there was slight neck retraction. Morphia gr. $\frac{1}{12}$ was administered. At seven o'clock the following morning, respiration suddenly failed. Camphor by injection and oxygen inhalation with artificial respiration were given. Within an hour there was complete restoration of the respiratory function, but five hours later she had another failure of respiration and died.

At the post-mortem examination on the same day, the bones of the vault showed definite pressure erosion. The floor of the third ventricle bulged forward into an enlarged pituitary fossa, and the pituitary gland was flattened and lay against the upper part of the anterior wall of the fossa. There was marked internal hydrocephalus and a large cyst of the vermis of the cerebellum. The absorption of bones on the floor of the middle fossæ was well marked. There was a spherical bulging behind the optic chiasma.

Discussion.—Mr. HUGH CAIRNS said that in making this freely acknowledged error of diagnosis, Mr. Graham Brown was in good company, for practically every neurological surgeon, at some time or other, committed the mistake of exploring the pituitary fossa in a case of midline cerebellar tumour. The mechanism by which the false localizing signs were produced was clear. In all cases of hydrocephalus, particularly when due to tumour of the vermis, there was a bulging of the third ventricle on to the roof of the sella turcica. At necropsy, in cases of acute hydrocephalus, one would see behind the stalk of the hypophysis a sharp depression in the diaphragma sellæ. Continued pressure in this region resulted in dilatation of the pituitary fossa, which simulated, radiographically, the enlargement produced by hypophyseal tumour.

False localizing signs were not limited to radiological appearances, but also occurred in the endocrine disorders. Just as menstruation failed to occur in Mr. Graham Brown's patient, so in older women amenorrhœa was one of the earliest symptoms. He (Mr. Cairns) had recently been asked to see a girl who was supposed to have a pituitary tumour. The girl had amenorrhœa, headaches, loss of vision and enlarged sella turcica, and this combination had led to a diagnosis of pituitary tumour. However, there was a history of vomiting at the onset of the illness, and on examination generalized hypotonia (rather more marked on one side than on the other), slight unsteadiness in walking, and a high degree of papillœdema, and in spite of the absence of gross cerebellar dysfunction he had made a diagnosis of tumour of the vermis and had explored the posterior fossa. On splitting the vermis he had found on the right side of the fourth ventricle a tough vascular glioma, part of which he removed. The fact that the menstrual periods had reappeared three months after the operation could be taken as evidence that the amenorrhœa was produced by the hydrocephalus, for, at operation, although only a part of the tumour had been removed, the obstruction to outflow of the cerebro-spinal fluid had been, for the time at least, completely relieved.

In cases of cerebellar tumour in which there was a long history, obesity was a common feature, and if papillœdema had been allowed to go on so long as to result in subsidence of the swelling, with optic atrophy and blindness, the resemblance to pituitary tumour might be still more close than in the case which Mr. Graham Brown had described. Diagnosis was not made easier by the fact that suprasellar tumours occasionally simulated cerebellar tumours very closely.

It was usually possible to distinguish radiologically between the enlarged sella turcica of pituitary tumour and the enlargement that was due to hydrocephalus. In hydrocephalus the

destruction of bone was usually confined to the clinoid processes and the dorsum sellæ, whereas in pituitary tumours it was in the floor of the sella that the destruction of bone was greatest. The visual fields were of great value, especially when measured with small visual angles. Vomiting in the early stages suggested strongly that the tumour was cerebellar rather than hypophyseal.

CASES.

Chronic Suppurative Otitis Media with Lateral Sinus Thrombosis and Cerebellar Abscess.

By L. GRAHAM BROWN, F.R.C.S.

January 8, 1926.—Male, aged 17, complaining of right otorrhœa of 2-3 years duration. Large polypus removed and cleansing treatment ordered.

February 4, 1926.—Admitted to hospital for removal of tonsils and adenoids. Aural discharge lessening.

July 22, 1926.—Admitted again to hospital as emergency case of acute mastoiditis. Rigor occurred soon after admission. Diagnosis of lateral sinus thrombosis made. Operation: extensive removal of necrosed bone, extradural abscess found with gangrenous lateral sinus thrombosis. Internal jugular vein divided between ligatures in neck, healthy lateral sinus exposed far backwards towards occiput and plugged. Wound partly closed in horizontal portion of incision but remainder left well open.

July 24, 1926.—Rigor occurred, then swinging temperature until August 13, 1926, when wound was reopened, pus and granulations removed and radical mastoid operation completed.

August 23, 1926.—Evening rise of temperature continued, with slight irritating cough. Chest and sputum examination negative, but X-ray report typical of a tuberculous involvement of apex of left lung.

August 27, 1926.—Patient complained of frontal headache on moving, and vomited several times but not independently of meals.

September 1, 1926.—For several days previously, temperature irregularly subnormal, pulse slow and full, vomiting and headache, much wasting and drowsiness.

Examination of central nervous system, including cerebellar functions: nothing definitely abnormal found.

September 6, 1926.—Symptoms of cerebellar abscess now definite. Operation: wound which had now almost healed was reopened and cerebellum explored with sinus forceps in site of the horizontal limb of the obliterated sigmoid. Immediate gush of pus about half an ounce. Rubber drainage-tube inserted without further disturbing surrounding parts. Wound left open.

September 11, 1926.—Marked recovery from cerebellar symptoms occurred from this date and no sequelæ remained.

January 16, 1928.—Slight moisture of mastoid cavity.

Discussion.—Mr. A. R. TWEEDIE said he noted that the tympanic membrane was still present, and that there appeared to be a suppurative lesion in the anterior part of the attic.

Dr. KERR LOVE said he thought the tympanic membrane was intact, but there was a discharge in the middle ear, and perhaps also from the antrum. He thought the intra-tympanic syringe should be pointed upwards and backwards, and the middle ear and antrum cleansed. Then there would be healing, unless there was discharge from the upper end of the Eustachian tube.

Severe Aural Vertigo Treated by Superior and Inferior Vestibulotomy.

By E. D. D. Davis, F.R.C.S.

A MARRIED woman, aged 33, incapacitated by aural vertigo and tinnitus of some months' duration.

In 1914 a radical mastoid operation was performed on both ears by Mr. Yearsley at the Royal Ear Hospital.

Both ears had healed perfectly, but there was very slight moisture of the Eustachian tube on the left. No nystagmus. No fistula phenomena; a tendency to fall to left.

Patient very deaf, voice heard at 2 ft. only from each ear. Bone conduction normal. Both labyrinths equally active to the irrigation test. The hearing of each ear appeared to be equal. Lumbar puncture: normal tension, normal fluid. No effect on the vertigo.

No cause for the vertigo could be found, apart from the ear condition. All kinds of palliative treatment tried.

November 17, 1927.—A right superior and inferior vestibulotomy with curetting of the vestibule carried out. The operation was easily and quickly performed. After operation, vomiting was excessive, with a riotous rotatory nystagmus in all directions, but more marked on looking away from the right (operated) ear. The nystagmus became much less in a few days and could only be obtained on looking to the left.

Pulse and temperature remained normal throughout.

Patient left the hospital after three weeks with no improvement in the vertigo and tinnitus. Gait very unsteady.

February 23, 1928.—Patient readmitted; a left superior and inferior vestibulotomy with curetting performed.

No post-operative vomiting, slight nystagmus on looking to the right.

April 3, 1928.—Patient much better and appears to have derived considerable benefit from the second operation.

The steps of the operation can be demonstrated on the temporal bone shown.

Discussion.—Dr. J. S. FRASER (President) said that from these cases one could learn the value or otherwise of operations on the inner ear in patients suffering from tinnitus and vertigo. His own limited experience was that the tinnitus cases were certainly better left alone. It was still a debatable matter whether the vertigo cases should be operated upon, except in a few well-recognized conditions, such as labyrinth fistula and tumour of the eighth nerve. The diagnosis of disease of the otolith apparatus, hypertension in the labyrinthine fluid, dilatation of the saccus endolymphaticus and serous labyrinthitis in the lateral cistern was still a matter of great difficulty.

Dr. ALBERT A. GRAY said he would be averse to operating on the middle-ear with the object of curing tinnitus, though it might be done for giddiness. Tinnitus was frequently of central origin; operation on the middle-ear and cochlea did not always cure it.

Mr. E. D. D. DAVIS (in reply) said it was difficult to find records of operations of this kind. Mr. Lake's view was that they were of no use. He (Mr. Davis) had been asked why the auditory nerve could not be destroyed to relieve severe tinnitus. Hence this attempt. The

patient suffered very much from vertigo, complaining but little of tinnitus, and he (the speaker) thought it a favourable case. There was improvement for a time, but the symptoms had returned and now there was more tinnitus.

It was easier to decide upon the operation in these cases because the radical mastoid operations had been done and the wounds had healed satisfactorily. The operation was easy; it took less than twenty minutes. Strict antiseptic and aseptic precautions were taken at the operation and during the dressings.

The Use of Temporal Muscle Grafts in the Radical Mastoid Operation (with Illustrative Cases).

By HAROLD KISCH, F.R.C.S.

AFTER the completion of the radical operation, a portion of the temporal muscle is separated partially from the main mass of the muscle. The fascia over the graft is divided, and some of the fibres cut across, until the graft can lie, without tension, in the cavity made at the operation. The meatus is then replaced, without any plastic operation. The skin incision is completely closed, and a plug placed in the meatus.

The graft attaches itself to the bone, and no further attention is given to the mastoid. The meatus and middle-ear are cleansed daily with peroxide drops and weak perchloride of mercury solution.

The patients are able to return home about a week or ten days afterwards and there is only a minimum amount of discharge from the middle-ear. After-treatment is painless, and hearing results are good.

Discussion.—Dr. J. S. FRASER (President) asked what was the object of this temporal muscle graft. He himself liked to make as large a meatus as possible, to line the cavity with a skin graft and get it dry, white and smooth. One could not always achieve such perfection but that was his aim. He did not see much point in filling the mastoid cavity with a piece of temporal muscle.

Mr. A. R. TWEEDIE said he understood that these cases had been only recently operated on, but at present there appeared no tendency for an epithelial covering to form over the graft.

Mr. NICOL RANKIN said that if successful the value of this method would lie in the time and number of dressings saved in the healing of the mastoid cavity. He would be glad to hear the reasons for doing it, and to see the cases at a later date.

Mr. KISCH (in reply) said radical mastoid cases required much attention and dressing afterwards, and he thought that if the cavity were obliterated the need for so great attention would be obviated. At first he used a portion of the rectus femoris muscle, splitting the meatus and then applying the graft. That was three years ago, and the cases did well. In only one instance did the muscle graft die, needing the reopening of the wound to remove it. Then it occurred to him it would be better to use a muscle which could be left attached by its base, and he chose the temporal muscle. In order to obtain this graft there was no necessity to enlarge the mastoid incision; it was easy to expose a large portion of the temporal muscle, and, by an incision through the muscle, to bring it down, so filling up the mastoid cavity.

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The question had been raised as to whether the muscle remained alive when it had been used. He had had the opportunity of re-opening a case and examining the graft afterwards, and he had found the mastoid cavity filled with the muscle, which was active, and contracted on stimulation. There was no necessity for a plastic operation on the auditory meatus, and the after-treatment was quite painless, whereas after an ordinary splitting of the meatus there was always some discomfort attending the dressing. The attic region was the only part which had yet to heal, but the amount of discharge from that area was very slight, and healing proceeded well.

Section of Dermatology.

President—Dr. J. M. H. MacLeod.

June 21, 1928.]

CASES.

(?) Dermatitis Vegetans.

By G. B. Dowling, M.D.

This, I am afraid, is a rather disappointing case. The patient, a female, came to me six months ago with an eruption on the legs, consisting of grouped vesico-bullous lesions, which I took to be infectious eczematoid dermatitis. I wrote to her doctor suggesting treatment, but the patient came back again two months later with the lesions enlarged and transformed into vegetating plaques. I found afterwards that she had been receiving bromide through other channels, and therefore we must assume it is a bromide eruption.

Dr. J. M. H. MacLeod (President) said he had recently treated a vesico-bullous eruption in a man following a dog bite. The eruption was so acute as to suggest acute pemphigus, and was accompanied by high temperature. The bacteriological examination was negative, but anti-streptococcic serum was prescribed; recovery took place after two months' stay in hospital.

Annular Sarcoid.

By G. B. Dowling, M.D.

Patient, a nurse, aged 44, whom I showed at the October meeting as a case for diagnosis. There is a perfectly oval lesion on the right leg, measuring 2 in. by 1½ in. The condition began twelve or thirteen years ago as a small palpable lesion in the leg. It gradually spread centrifugally, clearing up in the centre, and leaving scar tissue behind. The peripherally spreading edge has always been the same as it now is, that is, a slightly raised, yellowish-pink margin, over which can be seen large numbers of minute vessels. Section of the edge shows an infiltrate in the corium of large numbers of epithelioid cells and a fair number of giant cells, while a marked amount of fibrous tissue is being formed at the same time. It would appear that a tuberculous process is going on, which is being destroyed as rapidly as it is being formed, or almost as rapidly. The diagnosis seemed to me to rest between lupus and a very unusual sarcoid. I do not think it is lupus, because where it has healed the healing is complete, and there are no "apple-jelly" nodules to be seen, nor are there any fresh lesions in the scar tissue. I thought it bore some

resemblance to a case which Dr. O'Donovan showed here at the last meeting, and which he diagnosed as lupus.

Discussion.—Dr. W. J. O'DONOVAN said that the circinate superficially scarring infiltration shown by Dr. Dowling was of great interest. It differed from his own case (shown by Dr. Brain and referred to by Dr. Dowling) in several particulars. His (Dr. O'Donovan's) case was that of a female who showed a multiplicity of facial lesions, as in the case exhibited to-day; these lesions were circular and showed superficial central scarring. But the peripheral areas consisted of a coalescence of miliary, yellowish, slightly raised nodules. This present case was almost a replica of one now in hospital with similar leg lesions, from which there had been cultured a yeast-like organism, without mycelium.

Dr. A. M. H. GRAY asked whether Dr. Dowling had investigated this case from the point of view of Schaumann's other signs of lymphogranulomatosis benigna, as to tuberculin reactions or changes in the bones, as revealed by X-rays or changes in lymphatic tissue. In view of Schaumann's work, some of those indications should be obtained before applying the term "sarcoid." So far as the clinical and the microscopic evidences went, the case seemed to resemble the atrophic type of cutaneous sarcoid more than anything else.

Dr. W. J. O'DONOVAN (in reply) said that if the condition were proved to be tuberculous, or to be very probably tuberculous, the term "tuberculosis cutis," with or without qualifying adjectives, might be employed. If, however, the diagnosis could not be explained beyond the stage of innominate granuloma, he himself would prefer such a description to the term "sarcoid." Ambiguous terminology should be restricted, as it was reminiscent of times when a nomenclature such as lupiform syphilis was current.

Ear Condition in a Case of Raynaud's Disease.

By G. B. DOWLING, M.D.

PATIENT, a male, aged 26. During the last twelve years a mass of glands has persisted on either side of the neck, and some of these have been taken out from time to time during this period. On each occasion the pathologist has reported them to be tuberculous. Two or three years ago he developed Raynaud's disease, and at about the same time he developed the condition of the ears from which he now suffers. The lesions begin as red infiltrated papules, which become necrotic. The result has been the partial destruction of the lobes of both ears. I have never seen a tuberculide like it before, but the fact that it is so has been proved by Dr. Elworthy, pathologist to the West London Hospital, who has examined sections of the tissue and has pronounced them tuberculous. The distribution on the ears is obviously connected with the Raynaud's disease, but whether the Raynaud's disease has any relation to the swollen glands I do not know. It was suggested to me by Dr. Ironside that the enlarged glands in the neck might have been responsible for the Raynaud's disease by their involving the sympathetic; he showed me a case in a woman who had some large sarcomatous masses in the neck associated with Raynaud's disease of recent development.

Mycosis Fungoides.

By S. E. DORE, M.D.

PATIENT, a male, aged 49, who gives a rather vague history of persistent, slightly itching, scaly, circumscribed psoriasiform patches localized to the wrist and forearm, which are said to have existed sixteen years. Four months ago he received a slight injury to the front of the wrist, and this developed into a large proliferative ulcer. A microscopical section was made from the edge of the ulcer and the following is Dr. Braxton Hicks' report:—

"The epidermal papillæ and epidermis are greatly thinned out and beneath is an intense infiltration with cells of mesoblastic type and variable shape. These cells are spindle-shaped and of young fibroblast type, also lymphocytes and large mesoblastic cells. While it is possible that the condition comes under the heading 'inflammatory' there are strong suspicions that it may be early mycosis fungoides."

Though this must be regarded as an unusual case even of mycosis fungoides, the appearances seem to point to that diagnosis, and I think the evidence of the sections is consistent, if not conclusive. He has had three applications of X-rays, and the ulcer has flattened out and has decidedly diminished.

Discussion.—Dr. A. M. H. GRAY said it would be interesting if Dr. Dore could remove a piece of non-ulcerated tissue for microscopical purposes. The section was complicated as it was taken from near the edge of the ulcer, and from that piece of tissue diagnosis would be difficult. The case was unusual for mycosis fungoides, but he had no alternative diagnosis; it was extraordinary for that disease to be localized for so long.

Dr. J. M. H. MACLEOD (President) said that he could not put the granuloma in the section in any category other than mycosis fungoides. He did not think it was tubercle or syphilis; the irregularity of the cells suggested mycosis.

Case for Diagnosis.

By S. E. DORE, M.D.

THIS patient, a woman aged 26, presents on the right forearm, near the elbow, on the back, hips, outer surfaces of the thighs, right leg and neck, ringed patches of various shapes and sizes, with thick, raised spreading edges, some forming complete circles, others segments of circles, and some having incurved edges. They are stated to last about six weeks, are accompanied by very slight itching, and in certain areas by some amount of pigmentation. The patient said that she noticed the first patches on the right forearm three and a half months ago, and that new ones are continually forming. She has had no serious illnesses, but she had a bad miscarriage at three months two years ago. All her teeth were extracted a year ago. Wassermann reaction negative. The section from one of the patches showed only slight irritative round-celled infiltration about the capillaries, but otherwise nothing characteristic. I put the case down for diagnosis, as I had hoped to elicit views as to the nature and correct nomenclature of the eruption, but it probably comes under the erythema multiforme group, and has been described as erythema gyratum or centrifugum perstans. The President (Dr. MacLeod), and Dr. Gray, have observed similar cases recently.

Discussion.—Dr. A. M. H. GRAY said this case closely resembled that of a patient he had shown at the Section a year ago.[1] There were somewhat similar lesions on the outer side of the left thigh, except that the margin was more raised, and wider. It was the size of the index finger. [Dr. Gray showed the illustration of the case in the *Proceedings.*] When he saw that case he admitted he was not prepared to make a diagnosis, and none of the Members then present could say more than that it probably belonged to the group of persistent gyrate erythemata, of which many examples had been shown. For many years he had maintained that these cases were intermediate between erythema multiforme and granuloma annulare, and were related to Crocker's erythema elevatum diutinum. His case was the most extreme he had seen, and when Professor Jadassohn was here last year he (the speaker) arranged for him to see the condition; the Professor said he had never seen a case quite like it, and thought it might be an anomalous example of the Darier-Roussy sarcoid. The section was striking: it was packed with giant cells, with a few other inflammatory cells. The section of Dr. Dore's present case showed, however, practically no giant cells; it was almost wholly a round-celled

[1] See *Proceedings*, 1927, xx (Sect. Derm.), p. 114.

infiltration. But he (the speaker) did not think too much stress could be laid on this; it was due to the stage at which the affection happened to be seen. He did not doubt that these cases were closely related to the granuloma annulare group, of which many different types were seen.

Another patient whom he had shown some years ago had circular lesions at every menstrual period, disappearing in two or three weeks, then re-appearing almost at once. In most of the cases there was not marked pigmentation as in this case, nor the curious incurving of the edges as in Dr. Dore's and his own cases. He did not know what the ætiology was.

Dr. J. M. H. MACLEOD (President) said that the case reminded him of one recently under his care. The rings and gyrate lesions were as marked as in the present case. The patient was a man who, when seen, had had the eruption for five weeks. No cause was ascertained; he was given calcium lactate and the eruption cleared up in three weeks.

Dr. H. C. SEMON said that he had a clinically similar recurrent case in a young woman. She differed from the President's and Dr. Dore's patients in exhibiting severe constitutional and febrile symptoms with every recurrence of the ringed eruption, which had always been regarded as a sub-type of erythema multiforme. Benefit resulted from enucleation of the tonsils, but the attacks had persisted in a less acute form since 1913; a focal cause, possibly intestinal, was probably still present.

(?) Morphœa or Tuberculosis Cutis.

By W. N. GOLDSMITH, M.D.

K. S., MIDDLE-AGED married woman, came to Dr. Gray's out-patient department in March, 1927, with the present lesion on her left shin. It was diagnosed as morphœa by Dr. Gray and described as an irregular, dark brownish yellow patch, very hard. The upper edge showed hypertrophy and lower part atrophy. It had existed for three years and began after an injury in two patches, one above and the other below the injury, and these patches coalesced. Since then the extent of the lesion has slightly increased, and there has been no material response to treatment.

The main characteristic of the lesion consists in the skin being hard and firm, very smooth and shiny, brownish lilac in the thickened parts and yellowish in the depressed parts. One obtains the impression that the changes are mainly in the cutis. During the last few months a similar patch has appeared on the right leg. There is no tendency to ulceration and biopsy incisions heal up rapidly. She has suffered from indigestion for many years, also from rheumatic pains: a radiogram reveals slight osteo-arthritis of her lumbar spine.

Biopsy of a thickened portion of the edge, April, 1927.—Massive infiltration, mainly of small round cells and fibroblasts, involving the whole thickness of the skin and of the superficial part of subcutaneous tissue. The epidermis is thin, but otherwise fairly normal. Now, according to Kyrle, in scleroderma the cellular infiltration is always confined to the neighbourhood of the slightly dilated vessels and never attains to the extent of a diffuse flooding of the cutis. This may, however, occur in the condition known as idiopathic acrodermatitis atrophicans, some examples of which may clinically resemble scleroderma. But the difference between the two is that in acrodermatitis atrophicans the elastic fibres disappear early and the collagen is not obviously affected, whereas in scleroderma the collagen exhibits swelling and clumping, and the elastic fibres are normal. In this first biopsy from my case there is a marked disappearance of elastin from the infiltrated area, and not, I think, a very striking change in the collagen.

Biopsy, March, 1928.—Thickened lump at upper extremity. Epidermis normal. Deeper layers of cutis and subcutis contain large masses of small cells and giant cells arranged in tubercles. Mr. W. G. Barnard considers it a perfect example of a tuberculous reaction. The collagen bundles appear normal and take on a good red stain with van Gieson's method. In scleroderma they generally stain brownish. No tubercle bacilli found. I do not think such a histological picture has been described either in morphœa or in acrodermatitis atrophicans.

Biopsy, May, 1928.—From edge of new lesion on right leg. Half was injected into a guinea-pig by Dr. Hedley Wright. The other half was examined histologically. Here the epidermis is thin and the papillary body absent. Otherwise there is nothing to be seen in the cutis except thickening of the deep vessels. But deep down in the subcutaneous tissue, at the extreme edge of the section, is a well-defined little tubercle containing a giant cell. The state of the guinea-pig is so far quite normal, but only three weeks have elapsed since injection.

The *tuberculin reactions* of the patient carried out intradermically are positive, but only moderately so.

The histology of this case appears to assign it a different category from that of scleroderma; probably it should be classed with tuberculosis cutis, but, clinically, it is typically morphœa.

Discussion.—Dr. H. MacCormac said that in 1922 he and Dr. Stowers had exhibited cases at the Section with lesions exactly similar to those now present in Dr. Goldsmith's patient. In his case the pigmented scar on the leg, which was finely grained by numerous blood-vessels, was of twenty years' duration and resembled lupus erythematosus of the scalp. There was also a small hypodermic nodule in the right thigh. The opinion he then expressed was that the condition represented a form intermediate between lupus erythematosus and Bazin's disease.

Dr. J. M. H. MacLeod (President) said that he did not lay much stress on the presence of giant cells in the diagnosis of microscopical sections and did not know of giant cell formation in scleroderma.

Dr. A. M. H. Gray said that though one spoke familiarly of the hypertrophic and atrophic phases of scleroderma, especially in the diffuse type, he did not think, however, that one often saw the hypertrophic phase in localized scleroderma. He had seen a certain number of cases of morphœa, but he did not remember one in which this definite hypertrophic phase was present. It might be that as it was not often seen there had been no opportunity of cutting sections at that stage. It was true that, in the present case, one section was cut from a patch which showed atrophic changes, but the patch excised intact showed no atrophic change. The sections threw an interesting light on the possible ætiology of the condition. Whether by looking at a section only one could say that a condition was tuberculous he did not know. Apparently pathologists were prepared to state this, but he thought most Members of the Section would be disinclined to say, from the histological appearance alone, that a condition was due to tuberculosis.

Section of Medicine.

President—Dr. HUGH THURSFIELD.

[*April* 25, 1928.]

The Rôle of the Kidney in Non-renal Disorders.

By I. SNAPPER, M.D.

(*Professor of Medicine, University of Amsterdam.*)

UP till recently the kidney has been considered to be mainly an excretory organ. This opinion has prevailed in clinical pathology, probably because in nephritis the principal symptoms are caused by impairment of excretion.

The kidney has, however, certain non-excretory functions, as proved, e.g., by the recently discovered ammonia formation in this organ (Nash and Benedict). This ammonia formation is most needed when great amounts of acid have to be excreted in uræmic and diabetic acidosis. It must be emphasized that, from time to time during diabetic coma, the ammonia content of the urine is hardly increased, and it has been proved conclusively that during and after diabetic coma the excretion, and therefore the formation, of ammonia has been impaired. As ammonia is necessary for the removal of diabetic and β-hydroxybutyric acid from the organism by the urine, we here possess one example of impairment of a non-excretory renal function which is of clinical importance.

Analogous circumstances may influence the cause of uræmic acidosis.

Another non-excretory renal function was discovered fifty years ago by Bunge and Schmiedeberg, namely, the synthesis of hippuric acid by the kidneys of different animals. I have been able to prove that the human kidney also can effect this synthesis, and also, that another substance with some resemblance to hippuric acid can be formed by the kidney. Further, I have also shown that this function is not limited to synthesis, as the kidney may also bring about difficult oxidations. I shall not go into detail regarding the oxidation of complicated aromatic compounds by the kidney, because this is mainly of physiological interest, but I want to lay stress upon the fact that substances of great importance in clinical medicine disappear in the kidney, i.e., the ketone bodies, diacetic acid and β-hydroxybutyric acid. These poisons are formed by the liver but not destroyed, or scarcely at all, by this organ. On the contrary, the kidney is the organ in which the destruction of these obnoxious substances takes place. In the clinical application of those facts one must look, in cases of diabetic coma, to see whether the kidneys are in any way at fault.

Most patients with diabetic coma are cured by insulin, but there is also a group, fortunately much smaller, in which insulin is only partially effective. In practically all cases of coma, hyperglycæmia is present and the blood and urine contain large amounts of acids. If insulin is given, the blood-sugar returns to normal, and ketone bodies can no longer be discovered in the urine and the blood. While in most cases of coma consciousness is restored by this treatment, a certain number of patients, even if hyperglycæmia and ketosis disappear, remain drowsy. Of these patients a great proportion die in four or five days. It seems to me that in these

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cases the kidneys must play an important rôle. Clinical observation has always pointed to an influence of the kidneys on diabetic ketosis. We know first, that in diabetic acidosis and coma albuminuria is often found, and that frequently the amount of albumin is parallel with the degree of ketosis, increasing and disappearing with it. Secondly, it has been shown that a common signal symptom of diabetic coma, often indicating the approach of coma, is the presence in the urine of very peculiar casts, first described by Külz. When ketosis is present and many casts appear suddenly in the urine, diabetic coma is likely to develop in a short time. There is another symptom discovered only during the last few years, which also indicates impairment of the kidney during coma. In the blood-serum non-protein nitrogen is found in such amounts, that in some cases it is doubtful whether the diagnosis should be diabetic or uræmic coma. It has been said that abnormal nitrogen metabolism is the cause of this increase of non-protein nitrogen in diabetic coma, but Begg has shown that in these cases the urine contains only small amounts of nitrogen; evidently the high amount of non-protein nitrogen in the serum is an indication that the function of the kidney is impaired. At necropsy, after death from coma, the kidney is not found to be sclerotic; it is large and yellow, and has characteristic changes. The best-known change is the glycogen infiltration of the cells of the tubules, first described by Ehrlich, but there is other damage. Weigert has described not only the presence of this condition, but also coagulation necrosis of the cells of the tubules, and Armanni has described changes in the descending portion of the loop of Henle characteristic of "coma kidney." I wish to indicate the possibility that in these cases of diabetic coma not cured by insulin, although ketosis and hyperglycæmia disappear, death results from impairment of the renal function, caused by the injury which diabetes inflicts on the kidney. In the long run the fatty infiltration of all parenchymatous organs which occurs in diabetes injures renal function as well; this seems to me the reason why patients with diabetic coma cannot always be cured by insulin. If one remembers that the normal kidney can destroy the ketone bodies, it is possible that in a certain number of cases of diabetes, ketosis depends partially on the fact that the tissues indicated for destruction of ketone bodies cannot destroy them any more. I should like to make the following division of cases of diabetic coma: (1) those in which ketosis is brought about only by hyper-production of ketone bodies; even if destruction is completely normal, there may be such hyper-production that the whole organism is overwhelmed; if insulin is given in such cases, inhibition of production is effected and the patients recover from the coma; (2) those in which there is only slight hyper-production of ketone bodies, but the organs destroying the ketone bodies, for example the kidneys, are damaged. If insulin is given in those cases, of course the hyper-production of ketone bodies is inhibited and the ketone bodies disappear, but the organism remains intoxicated by other poisons connected with the insufficient function of the kidneys. The injury to the kidneys causes death.

If one realizes that the kidneys have so great an influence on the destruction of ketone bodies, one is enabled perhaps to understand another thing which has always been difficult to grasp. Petrén, of Lund, has shown that diabetes with ketosis can be treated effectively with high-fat diets. This is difficult to understand, since, as fat is the principal source of ketone bodies, one would suppose that patients with acidosis should avoid fat, and yet Petrén gives his patients large quantities of it. Petrén's diet, however, is not only characterized by large amounts of fat; there is also a moderate amount of carbohydrate, but (and I think this is the characteristic feature) extreme restriction of protein. If diabetes complicated by ketosis is treated with a large amount of fat, some carbohydrate, but hardly any protein, the patient does well as long as he remains in the hospital, but if he goes home

there is a great chance of his returning to hospital within two or three weeks in a state of coma. The patient tries to keep up the diet at home, but usually the extreme restriction of protein is not continued. The patient increases the amount of protein, but leaves fat and carbohydrate unchanged and severe acidosis develops. One has to look for an explanation of this elective action of protein on the metabolism of acetone bodies. Is it not possible that this extreme restriction of protein relieves the kidney, since the most difficult function of the kidney is the excretion of nitrogen? When larger amounts of protein are taken the function of the kidney is overtaxed and the destruction of ketone bodies is no longer possible. Therefore it is nitrogen excretion which affects the condition of the patient unfavourably, by the overwork this entails on the kidney.

I hope I have shown satisfactorily that the function of the kidney is not restricted to excretion. The kidney is able to synthesize and to oxidize, and for this reason has therefore an important influence upon intermediary metabolism.

I have endeavoured to apply these facts to the pathology of diabetic coma.

Discussion.—Dr. R. D. LAWRENCE said that what he had seen of the treatment of diabetic coma, especially in those cases in which insulin failed, led him to think that it was not so much the condition of the kidneys, but the degree of circulatory depression which was the essential point in determining recovery or death. True, in all the cases of coma that he had seen there had been protein and, usually, some casts in the urine, but the degree of this temporary kidney damage seemed to have little relation to the prospects of recovery, whereas he had never seen recovery in a case with a very feeble pulse and low blood-pressure. The anuric type with nitrogen retention was certainly very fatal, but here again the anuria seemed as likely to be due to low blood-pressure (which had been present in all those cases he had seen) as to serious kidney defect. Again, cases of diabetes complicated with chronic nephritis had not, in his practice, seemed specially liable to ketosis. Therefore his experience led him to think that Professor Snapper attached too much importance to the rôle of the kidney in the production of severe ketosis and coma in diabetes.

Professor Snapper attributed the ketosis which usually occurred with Petrén diets, when the protein was slightly increased, to an overstrain of the kidneys in dealing with a little more nitrogen, although the protein intake was still relatively low. He (the speaker) had had little experience of Petrén diets, which he regarded as unpractical, though experimentally interesting. In only a few of the Petrén cases could there have been defective kidneys, unable to excrete a little more nitrogen, and yet in most of them ketosis developed with a slight increase of protein, insufficient, it would seem, to overstrain them. Therefore he (the speaker) thought that in the intermediate metabolism of protein there must be some other more physiological explanation of the phenomenon than an overworked kidney.

He had been deeply stimulated by Professor Snapper's paper, and would be interested to have some quantitative data of the amount of ketones with which a healthy and diseased kidney respectively could deal, and the amount of ketones the kidney usually had to burn in different conditions of metabolism in normal subjects and in diabetics.

Dr. P. J. CAMMIDGE said his own observations and experiments had indicated that defects in the functions of the kidneys were more common in diabetics than was generally supposed, and than the presence of albuminuria indicated. He had long held the view that such defects were often an important contributory factor in the production of the coma met with in diabetes. The failure of insulin to control the condition in some instances, especially in old and chronic cases in which renal defects were most common in his experience, with the remarkably successful results obtained in others, more particularly in young diabetics in whom renal changes were comparatively rare, tended to support that view. Hitherto he had assumed that when insulin failed, the persistence of the symptoms was due to the effects of retained nitrogenous waste products, and there was experimental evidence that this might be the case. The suggestion made by Professor Snapper in his paper that the kidneys normally possessed the power of destroying ketone bodies offered another explanation which was worthy of serious consideration, but it seemed to him (Dr. Cammidge) that the evidence so far was inferential and not very conclusive; he would be glad to know whether there was

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any definite experimental proof that the kidneys really had such a striking and hitherto unrecognized function,

Dr. F. PARKES WEBER said he understood that in cases of diabetic coma very low blood-pressure, when present, suggested an extremely bad prognosis. He wondered whether, in such cases, the use of methods for raising the blood-pressure (adrenalin, pituitrin), in addition to the administration of insulin, had been known to prevent the usual fatal outcome.

Sir JOHN BROADBENT said that Professor Snapper had called attention to the importance of ketonæmia in diabetes as opposed to ketonuria. He (the speaker) concluded, from what Professor Snapper had said, that, as in chronic interstitial nephritis, estimation of the blood-urea was of paramount importance in prognosis, so also in diabetes was the estimation of the ketone bodies in the blood. He would like to know what degree of ketone retention Professor Snapper had found in the blood in cases of diabetic coma, and what was the normal amount, if any, in the blood of a healthy subject. He would also ask what part the ammonia played in the elimination of ketone bodies. Was its function mainly to neutralize the fatty acids, and so aid in their elimination, and was deficient ammonia production of the kidney a contributory factor in the ætiology of coma?

He had seen albuminuria associated with glycosuria in Jews, and it did not appear to be a serious complication. Marked degenerative changes in the kidneys, however, were found, post mortem, in long-standing cases of diabetes. In view of the important functions of ammonia production and oxidation by the kidneys, described by Professor Snapper, it seemed clear that retention of ketone bodies, due to diseased kidneys, determined the onset of coma in such cases, and administration of insulin could not avert the fatal issue.

Section of Laryngology and Section of Otology.

COMBINED SUMMER MEETING, MAY 31, JUNE 1 AND 2, 1928.

LARYNGOLOGICAL SESSION, MAY 31, 1928.

Chairman—Mr. J. F. O'MALLEY, F.R.C.S.

Achalasia and Degeneration of Auerbach's Plexus.

By A. T. RAKE, F.R.C.S.

[ABSTRACT.

THE plexus was really a cell station for the vagus nerve-fibres. These nerve-fibres ran to the plexus, whence new fibres passed to the muscle-fibres. The function of the vagal supply was the twofold one of preserving the normal œsophageal tone and supplying the cardiac sphincter; it caused relaxation of the sphincter preceding the normal peristaltic wave which took the food into the stomach. Therefore any lesion of the vagal nerve-fibres would upset this relaxation of the cardia, which must take place before food could reach the stomach. Lesions might be proximal to Auerbach's plexus, or in the plexus, or distal to it. In some achalasic cases the plexus was found to be about twice its normal size, the whole of it being infiltrated with small round cells. In addition there were multiple vessels not seen in the normal Auerbach's plexus. In the chronic stage there were around the fibrous plexus large blood-vessels with thickened walls; the appearance was, indeed, so typical that it had been suggested that change was primarily vascular. The author, however, was strongly of opinion that the change was primarily inflammatory. In none of the many sections which he had examined were there any ganglion cells.

Discussion.—Mr. J. F. O'MALLEY (Chairman) said that it had long been felt that it was very difficult to explain the peculiar symptom-complex in this condition. All were familiar with the masterly presentation of the subject by Dr. Brown Kelly in the Semon Lecture two years ago, though he did not say more than that it was a neuropathic condition. The present paper seemed to carry the subject further towards a solution. He asked that the present discussion should centre round the pathological aspect; as to whether the condition was purely pathological, whether there was a physiological factor, or whether anatomical peculiarities helped the solution. He mentioned anatomy, because the structure of the upper œsophagus differed from that of the lower in the normal subject. Still, it was found, in many cases, that there were striped muscles which passed right down in the human œsophagus, as was commonly found in animals. Perhaps there might be some temporary reflex disturbance, as in other parts of the body.

Dr. ARTHUR F. HURST said that a century ago morbid anatomists noticed enormous dilatation and hypertrophy of the œsophagus, without finding an explanation in the cardia. As there was no obstruction physicians naturally sought a functional cause—a spasm at the sphincter. Fifteen years ago it occurred to him that spasm was not an adequate explanation. Though in a small number of specimens some hypertrophy had been found, i.e., in the cardiac sphincter, in the great majority, including Mr. Rake's nine specimens, there was no hypertrophy of the last inch of the œsophagus. The speaker once saw the cardiac sphincter during life. In one case his mercury tube was not effective enough, and he asked Mr. Rowlands to operate. Mr. Rowlands found, in the inch which corresponded

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to the sphincter, that the tissue was very thin indeed, but the whole sphincter appeared to be present. He then asked Mr. Rowlands to perform an operation like that for hypertrophic pylorus. Mr. Rowlands divided the longitudinal fibres, but his incision did not pass through the diaphragm. The muscle coat was so thin that the surgeon did not think he had got through it, and he actually cut through the mucous membrane, but it healed well and gave no further trouble. That was seven years ago, and the man had been well ever since; it was clear he was cured by division of the sphincter, in which there was no trace of hypertrophy. That seemed to be good evidence against the presence of spasm. Had spasm continued for so long it would surely have left hypertrophy. In most of the cases the mercury tube passed in with no obvious resistance, and there was no gripping during its passage. It corresponded with Hirschsprung's disease in the colon. When, in 1914, he first described these cases he said the condition was probably due to absence of normal relaxation of the sphincter, a relaxation which ought to occur at each peristaltic wave, and the term "achalasia" was the suggestion of Sir Cooper Perry. He (Dr. Hurst) had suggested that it would be found that this condition was due to some organic change in Auerbach's plexus, and the suggestion was correct. In every one of the nine cases Mr. Rake had examined there was a gross anatomical and pathological change in Auerbach's plexus, particularly at the lower end of the œsophagus. Eighteen cases of the condition had now been examined. Dr. Cameron had found the same changes, though he did not publish the fact; there were certainly changes in the normal activity of the cardiac sphincter.

Dr. BROWN KELLY said that so far as he was aware, Mr. Rake was the first to describe fibrotic changes causing destruction of the nerve cells in the plexus. In justice to his colleague, however, the speaker stated that Dr. Munro Cameron was the first to demonstrate these changes in cases of cardiospasm. The case upon which Mr. Rake's first paper was based was one of hypertrophy of the wall of the œsophagus without dilatation, and the patient had had no symptoms of cardiospasm. In the three cases examined and reported upon by Dr. Cameron there was pronounced dilatation and characteristic symptoms of cardiospasm. The results of this investigation were briefly mentioned and illustrated in the Semon Lecture (December, 1926).

If he were to attempt an interpretation of the pathological findings, he would say that the degenerative changes in Auerbach's plexus caused an incoördination of the myenteric reflex in the region concerned. In consequence of involvement of the nerve cells and their connexions, the transmission of stimuli was interrupted. Stimuli excited by the bolus of food might readily pass to the subjacent circular layer, but might not reach the distant longitudinal layer; consequently the former might contract, while the latter might remain inactive.

With regard to the affection described as "cardiospasm" and "achalasia of the cardia," he did not think that sufficient was known about the normal nerve processes at the lower end of the œsophagus to offer a satisfactory explanation of the clinical appearances and tissue changes found there. Dr. Hurst's theory, advanced in 1914, was proposed in 1888 by Meltzler (*Berl. klin. Wochenschr.*, 1888, p. 140), who attributed the dysphagia and œsophagectasia to a lack of relaxation at the cardia. At that time, and subsequently, it was regarded as unsatisfactory and little attention was paid to it. Many cases were now on record of hypertrophy of the cardia; he himself had had a case which was described by the late Professor Shattock; Dr. Munro Cameron had had another, and Dr. Irwin Moore had illustrated and referred to several collected from various sources.

[Dr. HURST: All written communications on the subject refer to Sir Humphry Rolleston's pioneer article in 1894, in Clifford Allbutt's "System of Medicine," in which Sir Humphry said it was due to want of relaxation and of normal peristalsis.]

Dr. BROWN KELLY stated that Sir Humphry Rolleston did not know of cases of cardiac hypertrophy, and that led him to adopt the non-relaxation theory.

Mr. J. F. O'MALLEY (Chairman) said there was a difference of opinion as to the name suitable for this condition. "Cardio-fibrositis" might be a good compromise. Different names did not help to a solution of the nature of the disability.

Mr. W. STUART-LOW said he had been mystified by the suggestions in the medical press as to the cause of this condition. More attention should be paid to the clinical side. Whatever the pathology might be, he had relieved sufferers greatly by using warm olive oil

as a lubricant to the œsophagus; the patients were often in a low and nerveless condition, and the rear of the pharynx was dry.

The leading symptoms were clearly explained by the evident desiccation of the mucous membrane, and the therapeutical test of the success of lubricants was sufficiently significant and conclusive.

Dr. HURST said Mr. Rake's idea was that the degenerative changes were probably secondary to a primary inflammation, and this primary inflammation might be due to various causes, the most common probably being an œsophagitis spreading from the mucous membrane. The mucous membrane recovered its normal state, but the spread of the inflammation caused a degeneration of the plexus. In the first case there was no evidence of œsophagitis, and it looked as if the infection were blood-borne; it might be spirochætal, or that following an acute fever.

Dr. BROWN KELLY said that Dr. Munro Cameron recently described a case of round-celled infiltration which extended from the mucosa to Auerbach's plexus, thus showing that the process might have commenced in that way.

Dysphagia due to Pharyngeal Paralysis.

By W. M. MOLLISON, M.Ch.

DIFFICULTY in swallowing is one of the common symptoms for which patients present themselves, but to find that difficulty due to paralysis of the pharyngeal wall is not common, an impression which is confirmed by reference to standard text-books.

However, during the last few years I have been fortunate in seeing several of such cases. The subject is mostly unpractical, for we cannot attack the cause nor treat the symptoms with much hope of success, and perhaps for that reason it has its fascination. To study the causation of pharyngeal paralysis the laryngologist must step outside the perhaps narrow confines of his specialty and invade those obscure and difficult territories in the domain of neurologists.

The pharyngeal muscles are supplied by the pharyngeal plexus, which derives its fibres from the glosso-pharyngeal vagus and the accessory portion of the spinal accessory nerves.

The glosso-pharyngeal nerve is almost entirely sensory, only supplying fibres to the stylopharyngeus, which is of minor importance in the movement of the pharynx during swallowing. The fibres supplying the pharyngeal plexus are derived from the nucleus ambiguus lying in the floor of the fourth ventricle. The diagram here reproduced (p. 80) illustrates the anatomical position.

A few statements here given from Bing's Compendium are perhaps in the knowledge of many Members, but they help to render the position clear so far as the nerves under review are concerned.

Complete unilateral paralysis of the vagus sets up unilateral paralysis of palate, pharynx and larynx, but the interference in deglutition is almost always trifling.

The laryngeal paralysis when complete causes the cord to become flaccid, and both adductors and abductors are paralysed. If the paralysis of the vagus is incomplete posticus paralysis only is seen.

The glosso-pharyngeal nerve is almost exclusively sensory; the stylopharyngeus is of so little importance that its paralysis causes no observable functional loss.

The medulla is so small in volume that an isolated nuclear paralysis of either the ninth, tenth, eleventh or twelfth nerve is hardly ever seen; this is borne out by the cases I shall quote.

A nuclear lesion of the twelfth nerve usually causes bilateral paralysis of the tongue, as the right and left nuclei are so close together; combined paralysis of the

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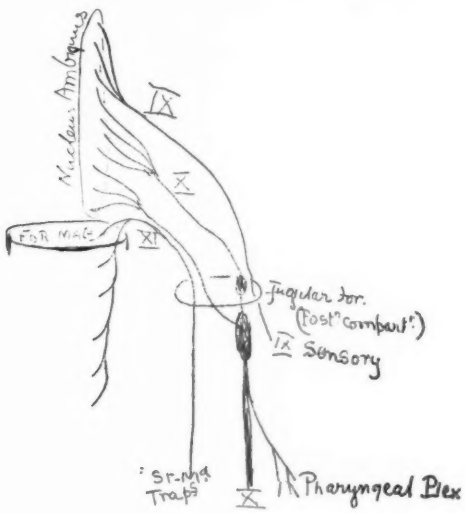
various nerves of this, the "caudal group," the ninth, tenth, eleventh or twelfth, may result from lesions which are supranuclear, nuclear or infranuclear, but also intracranial (tumours, aneurysms, exudates, etc., in the posterior cranial fossa). We are dealing mainly with nuclear damage.

After exit from the skull conditions necessary for simultaneous destruction of several of the nerves under review are very rarely found. Such lesion must be high up in the neck (two cases of this are known). Dr. Brown Kelly touched on this point in dealing with paralysis of vocal cord in his paper read last year.



Working backwards from the cases I have seen, I suggest that it is possible to imagine a small hæmorrhage occurring in the middle of the nucleus ambiguus. Such an hæmorrhage would damage the fibres to the pharyngeal plexus and thus the palate, pharynx and larynx; as the effects of the hæmorrhage became localized to its immediate vicinity the effects might be restricted to the palate, the pharynx or the larynx.

For the sake of the clinical aspect the causes of pharyngeal paralysis may be thus classified:

(*A*) Central or nuclear	... ...	Bulbar paralysis; localized hæmorrhage; embolus.
(*B*) Intracranial or infranuclear	...	Pachymeningitis; tumours.
(*C*) Extracranial, base of skull	...	Tumours; diphtheria, lead poisoning.

Classification of Causes.

(*a*) *Central.*—True bulbar paralysis or localized hæmorrhage.
(*b*) *Intracranial.*—Pachymeningitis, tumours.
(*c*) *Extracranial.*—Tumours, glands, F.B. base of skull.
(*d*) *Peripheral.*—Diphtheria, lead poisoning.

It might well be expected that cases of paralysis of the pharynx due to central lesions are not common; such I think is the case, and reference to them in the standard text-books would appear to support that view.

Reference to standard text-books on the nervous system makes it clear that there is a condition recognized as bulbar paralysis, a slow progressive disease in the medulla, of which dysphagia due to paralysis of the pharyngeal muscles is one symptom; but the impression obtained is that this disease always progresses and has a fatal ending. An acute bulbar paralysis is also described, a similar but more rapid affection (Beevor[1]).

I have been fortunate in meeting cases which do not appear to conform to this impression gained from the text-book. In six cases to be described five recovered, apparently permanently. In some of these cases severe straining preceded the onset of symptoms, in two from vomiting and in the third from whooping-cough; in all three dysphagia was the only marked symptom remaining after recovery of the patient, and it is possible that this straining caused a small hæmorrhage from which the patient recovered, but with localized nuclear damage.

Prognosis.

This is favourable in diphtheritic paralysis; unfavourable in progressive muscular atrophy and amyotrophic lateral sclerosis, death occurring within a year or two. In A. C. bulbar paralysis it is better, though a further attack may occur.

Case I.—Patient, a female aged 84, was seen with Dr. Mahon in June, 1925. She complained of difficulty in swallowing of four years' duration. Four years previously she had had whooping-cough, and the dysphagia had begun afterwards. She can swallow enough to keep her in health, but it is difficult. For lunch on the day of examination she had had soup and a lamb cutlet cut up finely; there was a good deal of mucus which she had to cough up; she had not lost weight. On examination there was much frothy mucus in the deep pharynx and about the arytenoid region, filling the pyriform fossæ; the pharyngeal wall did not move and a diagnosis of paralysis following whooping-cough was made. It is of interest to note that three years ago, i.e., one year after the whooping-cough, two most competent laryngologists had examined the patient, and had diagnosed neoplasm, probably of the post-cricoid space; the presence of the frothy mucus was exactly like that seen in such a condition. This patient was an active woman and was able to enjoy life in spite of her disability. Dr. Mahon wrote me in May, 1927, that the patient was still "going strong," and her difficulty in swallowing was *in statu quo*. She had had a bad attack of influenza and mild bronchitis, but made a good recovery.

This lady was seen again in December, 1927, and examination showed the same condition as two and a half years before.

It is suggested that during a spasm of coughing a small hæmorrhage occurred in the bulb; that this resulted in damage of the nucleus ambiguus of the bulb with resulting paralysis of the throat at the time of the whooping-cough; no doubt the patient was too ill; at the age of 80 whooping-cough is a dangerous disease. It is possible there may have been other muscles involved at first which recovered (*cf.* Case II).

Case II.—Mrs. B., aged 67, was seen with Dr. Napier Jones in March, 1927, on account of severe dysphagia of twenty-four hours' standing. On March 4, some twelve hours after eating some fish at lunch she was seized with diarrhœa and vomiting: the retching was very severe and she had a collapse while in the lavatory. She was helped to bed; she then found her voice was weak, and that she could not swallow. The patient, when seen twenty-four hours later, was in bed propped up on pillows, as that was the most comfortable position for her; she looked pale and feeble. Examination showed loss of movement of the right side of the soft palate and the right side of the pharyngeal wall; there was much frothy mucus collected about the deep pharynx and larynx; the right vocal cord did not move. There had been some question of difficulty in moving the legs, but all reflexes were normal. Nasal feeding was advised and was successfully carried out, though the passage of the tube was difficult on account of a septal spur. The patient could take sips of tea. When seen again a week later, the patient could get down a little food, but it made her cough. Afterwards recovery was steady, and about five weeks later she was able to come to London from the country. The

[1] "Paralysis of the Soft Palate," *Clin. Journ.*, 1896, Oct. 7, p. 376

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right side of the palate moved nearly normally; the right cord had almost recovered as well: but the pharyngeal wall remained paralysed. Swallowing was fair; but fluids still caused coughing, as also did certain other kinds of food. Upon examination four and a half months later, the voice was a little husky but the cords moved well, though the movement of the right was "nystagmoid." The palate moved normally but the pharyngeal paralysis remained.

The patient can swallow with fair success, but she has to be careful not to talk or laugh while eating. She notices a loss of sensation to heat and cold in the right hand though the power is perfect both in arms and legs. (Incidentally it may be mentioned that she has had a motor accident since the illness.) With the exception of the pharyngeal paralysis this patient has recovered. In this case also it is suggested that a small hæmorrhage took place in the bulb as a result of the retching with the involvement of the nucleus ambiguus of the nerves to the palate, pharynx and larynx, partial recovery following.

Case III.—Mr. B., aged 68, seen with Dr. Lewin in April, 1925. Seventeen days earlier he was suffering apparently from a bulbar hæmorrhage; this caused paralysis of the right side of the face, the palate, the tongue, the pharynx and vocal cord. His present difficulty consisted in swallowing.

Examination showed that there was recovery from the paralysis of all the higher muscles; the left arytenoid moved a little better than the right and the lower part of the pharynx was still inactive. He had the greatest difficulty in swallowing, but the passage of a bougie was of great assistance, and it is probable that there was a considerable functional element added to the organic dysphagia. The patient recovered.

> In April, 1928, Dr. Lewin wrote: "Mr. B. goes to business five days a week; he had influenzal broncho-pneumonia fourteen months ago, from which he recovered with difficulty. He cannot walk far, pulse 50 and irregular, arteries tortuous, blood-pressure 170, as it has been for some years. The swallowing has never completely recovered; if he takes plenty of time and soft food he is all right; if he swallows carelessly, a little 'goes the wrong way' and he coughs violently; on this account he takes his meals alone. I am sure his pharyngeal muscles have not quite recovered their orderly function. He sometimes finds pieces of green vegetable in his throat, perhaps after the next meal but one."

This patient is now over 71, and it is obvious from the foregoing account that he is really fairly active.

Case IV.—S. R., male, aged 56, was admitted to Guy's Hospital in May, 1927, under the care of Dr. Conybeare, on account of difficulty in swallowing. This dysphagia began twelve or thirteen days before admission; it came on after an attack of vomiting when he "strained very much"; the patient considered this vomiting followed a dose of "salts," taken for an attack of abdominal pain. During the straining he became dizzy and lost consciousness for a short time. Examination showed paralysis of the right half of the soft palate, of the right side of the pharyngeal wall, and the right vocal cord moved poorly. It is again suggested here that the severe vomiting and retching caused damage in the bulb, resulting in the clinical picture described.

Dr. Symonds examined this patient and reported as follows: "He has the characteristic symptoms and signs of blockage of a certain small vessel supplying the lateral part of the upper half of the medulla on the right side." He added the following, which seems of considerable interest: "The vessel affected is *not* the posterior inferior cerebellar, but a short circumferential branch of the basilar, which the French call *l'artère de la fossette latérale du bulbe*." Foix's first paper gives a beautifully accurate account of a case with autopsy. I have seen one or two before, but never one quite so clean-cut as this one.

Foix draws particular attention to the fact that though the palate and pharynx are severely affected the vocal cord is relatively spared, arguing from this that these fibres—to the vocal cord—arise from the lower part of the nucleus ambiguus in the lower part of the medulla.

The physical signs in the present case, which are perfectly clear, are the following:—

(1) *On the right* (the side of the lesion).—(*a*) Miosis and enophthalmos. (*b*) Blunting of all forms of sensibility over the fifth distribution. (*c*) Paralysis of soft palate and pharynx, cerebellar ataxy of the limbs.

(2) *On the left.*—Blunting of sensibility to pain, including the whole body below the trigeminal area.

This patient died at home on September 21, 1927. His widow wrote that "he had quite regained the use of his throat before he died."

Possibly this case should not be classed with the previous three, as his disease wa apparently progressive.

Case V.—E. D., female, aged 44, attended the out-patient department on account of "hoarseness," with the history of always being liable to colds and sore throats; that she had lost her voice eight months ago and had been husky on and off ever since. She used to have difficulty in swallowing.

Examination showed extensive nerve involvement; thus, the tongue was pushed out to the right and the right side was less firm than the left; the right side of the soft palate was paralysed, the side of the pharynx and the right cord paralysed; further, the right trapezius and sternomastoid were partly paralysed. The patient appeared perfectly well in other ways.

Dr. Wilson, of St. Albans, writes:—

> "I think her general condition has slightly improved—capacity, walking and doing light jobs—but she is worse in one or two respects. She complains of considerable pain round the right shoulder-girdle and along the right sternomastoid. She gets uncomfortable sensations in the right side of the tongue, sensation of twitching of right side of face and eyelids and feels as though the mouth is drawn to one side; there is actually no paralysis of the face but the right shoulder is lower than the left. The tongue, soft palate and larynx paralysis are much the same, if not slightly improved. She has occasional difficulty in swallowing liquids without regurgitation through the nose, and anything too sweet or with much taste in it irritates and produces a paroxysm of coughing. She is definitely losing weight. Wassermann negative."

Case VI.—Miss M. B., 63, seen, in March, 1924, with the late Dr. Brock, on account of difficulty in swallowing.

Ten weeks previously she had had "influenza" with a rise of temperature which persisted for two to three weeks; she recovered sufficiently to go for a holiday. Eight days before examination the patient had difficulty in swallowing followed by pain in the left parietal region and in the left ear, so severe one day as to necessitate an injection of morphia; the temperature had risen to 102° F., but had been normal now for two days. On one occasion she had vomited very suddenly without any nausea. Five days ago the voice "changed."

Examination showed the tympanic membrane to be normal; paralysis of the left side of the soft palate, left pharyngeal wall and left vocal cord, the left arytenoid was very red and on it was a white membrane covering an ulcer, the left pyriform fossa was full of frothy mucus. No glands felt in the neck.

Diagnosis.—Paralysis of the fibres from the nucleus ambiguus; it was tentatively suggested this was due to encephalitis (at that time prevalent), or to influenza. The pain in the ear referred of course to the inflamed arytenoid.

The patient recovered, though the difficulty in swallowing persisted and the left cord remained fixed. Unfortunately the subsequent history is unobtainable, but a friend reported that she had recovered.

This case is of interest, as the paralyses of the palate, pharynx and cord were discovered in the routine examination of the patient.

Case VIa.—Mr. H. N. P., aged 48, seen in June, 1926, on account of deafness and some vertigo on blowing nose or coughing.

Complete ptosis of left upper lid and loss of sight in that eye. Had been seen by Mr. Lawford. Left vocal cord found fixed. Wassermann reaction negative. Incidentally, hearing in both ears equal and fairly good.

Examined again December, 1927, on account of recent (two months) great increase in deafness of right ear. Examination now showed the tongue pushed out slightly to the left; left side of soft palate moved less well than right. Curtain movement of pharynx to the right. Left cord fixed (probably these signs were missed at the previous examination). Voice excellent, only coughing or deep inspiration betrays any symptoms of the left cord fixation.

This case is quoted as a probable example of a pharyngeal paralysis and paralysis of cord of long standing giving no symptoms. The following is an "embolic" case.

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Case VII.—Miss S., aged 41, seen on September 24, 1927. Ten days earlier patient was about to go upstairs to bed when she had a severe attack of vertigo, vomited, and appeared very ill. She was put to bed and continued to vomit all night; was somewhat confused mentally.

She had suffered from aortic and mitral valvular disease for years, and during the attack of vomiting her pulse became very irregular and feeble. She had much cough and a good deal of inspiratory stridor. Since the attack there has been complete inability to swallow and the patient has been fed by means of a gastric tube; this artificial feeding had to be continued for three weeks from the attack. Owing to embolic gangrene the left leg had been amputated above the knee some years before.

Examination showed the patient to be somewhat cyanotic: there were dilated venules over the malar prominences and chin: she only whispered, though she could cough well and brings up a quantity of frothy mucus.

Left side of palate flaccid; right moved normally; left side of the pharynx paralysed. Larynx very difficult to see exactly, as it was covered with frothy mucus, which patient could not get rid of. But as far as could be seen both cords moved and the fact that the patient coughed well helped to confirm that opinion.

Diagnosis.—Embolism into the bulb in the region of the nucleus ambiguus.

Later (November 20) her brother, a doctor, wrote that the patient died in her sleep, presumably from a further embolism; the power of swallowing had partially returned and for a fortnight before her death she was able to swallow with a little difficulty. The paralysis of the palate also remained; but the voice became quite normal and she ceased to suffer from any laryngeal irritation.

The following case was due to peripheral nerve paralysis.

Case VIII.—P. B., school girl, aged 11½, attended Out-patient Department in March, 1928, complaining of difficulty in swallowing and return of fluids through the nose. She had much difficulty in swallowing solid food. She was just recovering from what was diagnosed as bilateral quinsy.

Examination revealed partial paralysis of the right side of the soft palate, well-marked curtain movement of the pharyngeal wall, showing paralysis of the right half, and some saliva lying in the pyriform fossæ. The cords moved normally.

This case would suggest the presence of post-diphtheritic unilateral paralysis: against this view, however, is the fact that two swabs of the throat taken while the child's throat was acutely inflamed failed to show any Klebs-Loeffler bacilli.

There is no reason why the case should not be one of streptococcal neuritis; cases of palatal paralysis after streptococcal infection are known.

The following cases were due to polio-encephalitis.

Case IX.—A. G., a boy aged 14, attended in the Throat Department on account of difficulty in swallowing. Four days previously he awakened to find he could only swallow with difficulty; he could swallow if he took some fluid to wash the food down. He found he could not play a wind instrument; and during the last four days he had had difficulty in speaking clearly on one or two occasions. Examination showed paralysis of the right half of the palate; a great deal of mucus adherent to the posterior pharyngeal wall and paralysis of the right half of the pharynx. Dr. Symonds saw the patient and considered the condition to be due to polio-encephalitis; he gave a good prognosis.

Case X.—J. A., aged 61, railway signalman, attended Throat Department, October 3, 1927, on account of difficulty in swallowing. Two months earlier patient had got wet through, and shortly afterwards suffered from an attack of "neuralgia" over the right side of the face. Fourteen days after the wetting he suddenly found he had difficulty in swallowing; a month later he became hoarse. No pain in the throat. Examination showed paralysis of right half of soft palate, "curtain" movement on pharyngeal wall—paralysis of right side, and impaired movement of the right cord.

Dr. Symonds saw the patient and reported weakness of right trapezius and sternomastoid, and weakness, and perhaps atrophy of both sides of tongue. It was, he thought, probably a case of progressive bulbar paralysis.

Actually the patient made a perfect recovery in a few weeks and it may have been a case of mild polio-encephalitis.

I have been fortunate in seeing the following two cases in which the lesion has been at the base of the skull:—

Case XI.—Male, received gunshot wound during the war, the bullet entering below the right ear; X-ray examination showed that it was lodged at base of skull. Right side of palate, right side of pharynx and right vocal cord paralysed. Had the greatest difficulty in swallowing. The bullet had damaged the ninth, tenth and eleventh nerves as they emerged from the jugular.

Case XII.—Male, aged about 33, seen in the Out-patient Department on account of difficulty in swallowing of only two or three days' standing. Right palate and pharynx paralysed. A mass of tuberculous glands had been removed by Sir Charters Symonds twenty years before.

Dr. Symonds saw him and thought it probable that he had a tuberculous gland at the base of the skull, involving the nerves at their exit.

I realize that these cases are of academic interest only, but the fringes of a specialty have their importance, and certainly extend our interests.

Discussion.—Dr. D. R. PATERSON thought it would be well to add to such a collection cases of myasthenia gravis in which dysphagia was sometimes met with. He had seen two cases, both in women, who had considerable difficulty in swallowing though nothing local to account for it could be discovered. The tube readily passed into the gullet, and the report from the physician's side regarded them as cases of myasthenia gravis. There was probably some paresis of the lower part of the pharynx.

Dr. D. A. THOMSON said that he had observed for the past ten years a case of myasthenia gravis in which the patient was confined to bed entirely; the most prominent symptom was difficulty in swallowing. Several opinions were invited to exclude neurosis, the general opinion being that it was undoubtedly myasthenia gravis. Treatment by long-continued intramuscular injections of strychnine sulphate seemed to act best. She could now walk with care and enjoy the simpler pleasures of life to a limited extent, though there was always the grave fear of a sudden relapse.

Dr. LOWNDES YATES said that in most of the cases of the kind there appeared to have been some anæsthesia of the larynx; thus, mucus was not coughed up, but lay where it was secreted. One of his patients was given a bismuth meal, and it was passed straight down the trachea, there was no coughing and the diagram showed bismuth in the trachea and bronchial tubes. The sensory nerves would probably not be affected by a lesion of the nucleus ambiguus, and the question was whether the constant presence of mucus did not accustom the mucous membrane to its presence.

Mr. W. STUART-LOW said it seemed that the toxin of influenza was responsible for some of these cases. For the somewhat extensive paresis, strychnine, given both internally and intramuscularly injected, together with rest in bed, was of great assistance.

Dr. BROWN KELLY asked whether Mr. Mollison had met with old people who complained of dysphagia, but in whom no objective fault could be found. He had seen several cases of this kind, and had been puzzled as to the explanation. He also asked whether in paralysis following diphtheria the speech alone was affected when the palate only was implicated? but when there was also difficulty in swallowing, was the constrictor in addition involved? Also when there was bilateral recurrent paralysis of the cords, due to a peripheral cause, such as a growth involving both recurrent nerves, was dysphagia present? He thought there would be, because the nerve twigs to the upper part of the œsophagus were then involved.

Sir JAMES DUNDAS-GRANT said he hoped to bring to the meeting on the following day a young man who illustrated the points brought out in Mr. Mollison's paper. For some weeks he had had a speech difficulty, and had paralysis of the left half of his palate, following a severe tonsillitis. There was no paralysis of the vocal cord. When a cold mirror was placed under the nose during phonation air came only through the side of the nose on which the palate was paralysed.

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Mr. MOLLISON (in reply) said he had seen two cases of myasthenia gravis, though in one the diagnosis was in doubt. One, a man, had had dysphagia for four years; he recovered somewhat, then had an exacerbation and was sent to him (the speaker). He had paralysis of the soft palate, but no organic stricture of the œsophagus, and after the tube had been passed his state improved greatly. He had not advised strychnine, but after what Dr. Thomson said he would do so.

He had not gone into the question of anæsthesia in the cases mentioned in the paper.

Methods of Estimating the Liability to Post-operative Hæmorrhage from Unsutured Wounds.

By A. LOWNDES YATES, M.D.

SOME years ago I had a series of four post-operative hæmorrhages after tonsillectomy; these occurred although I was convinced that I had ligatured every bleeding point. In two of them it was a noticeable fact that the patients gave a history of spontaneous epistaxis.

In the same year I had several cases of spontaneous epistaxis in my Out-patient Department, and in a number of them bleeding could not be stopped permanently by the cautery. In one of them I found that the blood flowing from the patient's nose did not appear to clot; I therefore investigated the clotting-time of the blood in all patients with spontaneous epistaxis. I found that in three-fifths of these the clotting-time was prolonged from the normal time (three to six minutes) to fifty minutes in my most marked case. I gave calcium to reduce the clotting-time, but this had no effect. One patient was suffering from a condition in which acidosis was a feature, and I gave sodium bicarbonate. This patient had shown no alteration in his clotting-time with calcium, but with sodium bicarbonate the clotting-time returned to normal. I therefore tried the effect of sodium bicarbonate on the other cases of epistaxis, and I found that, if their clotting-time was prolonged, in the majority of cases, it returned to normal as soon as enough sodium bicarbonate had been given in order to make the urine alkaline. I also found that the liability to hæmorrhage recurred a month or so after the sodium bicarbonate was stopped. I then investigated a further group of cases to find out if this acid state was due to a focus of infection, for the results of another investigation had led me to believe that this might be so. I found that the clotting-time could be returned to normal, either by adequately dealing with the toxic focus or by giving sodium bicarbonate.

About this time I had a very instructive case from this point of view. The patient was a woman of thirty-two who had had one attack of epistaxis and required a tonsillectomy to remove a focal sepsis. Previous to operation the clotting-time was five minutes. The tonsils were removed under chloroform and ether, and she left the theatre free from hæmorrhage. Three days afterwards she had a secondary hæmorrhage, although the throat showed no evidence of well-marked sepsis. No bleeding point was visible, but both tonsillar beds were oozing blood. The clotting-time was thirty minutes and she had marked acidosis. She lost a considerable quantity of blood and the clotting-time returned to ten minutes. Two days afterwards the clotting-time had risen again to twenty minutes, and I then increased the dose of sodium bicarbonate, until in three days her urine became alkaline and the clotting-time returned to normal.

As a result of the peculiar features of this case I investigated the clotting-time in 143 patients presenting themselves for tonsillectomy with a history of epistaxis or with signs of acidosis such as a greasy skin, etc., or with evidence of marked sepsis in the nose, or nasal sinuses. I found that the relative alkalinity of the blood was normal in the twelve such cases which were tested. In the majority of cases the early morning urine was very markedly acid to litmus. If, in addition, the

clotting-time was prolonged, sodium bicarbonate was given in doses of one teaspoonful in a tumblerful of cold water, twice a day for adults, and a less quantity for children. The number of days for which the patient had to take the alkali before the urine became alkaline to litmus seemed to be a good working method of estimating the degree of compensated acidosis of the patient, and there seemed some connexion between the degree of compensated acidosis and the time of clotting of the blood; for, generally speaking, the clotting-time was prolonged roughly in proportion to the degree of acidosis compensated.

I must digress here to state that the term "acidosis" is misleading. I am informed that if the blood became acid for one second the patient would immediately die. The relative alkalinity of the blood in "acidosis" apparently does not change at all, but this is brought about by neutralization of any tendency to excess of acid by the bases of the blood or tissues. The term "compensated acidosis" would appear to be more accurate.

I then investigated a series of cases in which a toxic focus was present but in which there proved to be a normal clotting-time. Some of them had a highly acid urine which took as long to render alkaline with sodium bicarbonate as did those cases in which the clotting-time was much prolonged. It was therefore clear that the alteration in clotting-time did not depend upon the presence of every type of compensated acidosis, but only upon some special type. The reason for this difference is not quite clear; but a surmise which fits the facts is that the organisms in a toxic focus, when they lead to acidosis, elaborate acid products which have a greater affinity for some bases than for others, and that when an acid is elaborated with a great affinity for calcium it combines with the calcium of the blood which is no longer all available for activating fibrin ferment.

Whatever the theoretical consideration on which this question rests, the clinical fact remains that, in compensated acidosis where the clotting-time is much prolonged, it can be returned to normal by giving sufficient alkali to neutralize the urine as it is secreted.

Using this method of rendering the clotting-time normal previous to operation I have probably escaped trouble from post-operative hæmorrhage; but it goes to prove how unwise it is to feel secure when I state that in four years at the General Hospital, Wolverhampton, I had, until last week, no case of post-operative hæmorrhage after dissection of tonsils, some four of which I carry out every week.

Last week, however, I had a case of the very type in which one might expect a secondary hæmorrhage, namely, a man weighing 14 stone, with rheumatism and septic tonsils. I did not test his clotting-time; after operation the bleeding was furious on his return to the ward and, though we took him back to the theatre, no bleeding point of any size was visible. Suturing the pillars did not stop the hæmorrhage, it stopped only when the patient had lost sufficient blood to reduce his clotting-time to normal.

I have quoted two of my failures, and of several successes I will quote only two.

K. C., male, aged 20, had suffered from indefinite nervous symptoms and from an indefinite abdominal complaint for about fifteen months. He was sent to me for search for a focus of infection. The tonsils were extremely septic and I advised removal. He gave a history of two attacks of epistaxis, one three months previously. I tested the clotting-time and found it to be fifty-five minutes on November 30, 1927. He was sent home for treatment with the alkali. In mid-December his doctor reported that the urine was now alkaline and the clotting-time normal. He was re-admitted; the tonsils were removed by dissection with about an ounce and a half of hæmorrhage.

R. B., aged 5, had suffered from purpura hæmorrhagica and had markedly septic tonsils which I was asked to remove. His blood clotting-time was twenty minutes and if his ear was pricked it bled for the whole afternoon. His urine was highly acid, it took nearly a fortnight to make it alkaline with sodium bicarbonate. Tests of the clotting-time and

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bleeding-time were taken, and these gradually fell, and when the urine became alkaline the tonsils were removed and he lost altogether something under one ounce of blood (a blood donor was waiting to be of use in case of hæmorrhage).

There are many similar cases, and all of them go to prove that in a considerable proportion of cases with a much prolonged clotting-time, this can be returned to normal by giving massive doses of alkalies.

In certain cases this alteration of the clotting-time occurs in rickets, and it is possible that the rickets may be produced by some such call on calcium. This is clearly not the case in most patients suffering from rickets, for observations show that the calcium content of the blood is low in rickets, and hence it may be that this lack of calcium causes the tendency to bleed; moreover, cases with rickets fall into two groups in this connexion, that in which the clotting-time can be returned to normal by giving alkalies, and that in which it can be returned to normal by giving calcium. This liability to hæmorrhage in acidosis may be a factor in bleeding from unsutured wounds other than those of tonsillectomy; for example, it has been noted in one of my cases that the tendency to hæmorrhage disappeared when the toxic focus from the nose was dealt with and alkalies were given.

The simplest methods for testing the clotting-time are much the best. I prick the ear and put a drop of blood upon a slide. When this clots it will not run up and down the slide when this is moved. Examinations should not be made at intervals shorter than three minutes, as constant movement delays the clotting-time. They should be made at even temperature, if possible at 60° F., for if under 50° F. the normal clotting-time of normal blood is delayed. When the blood is clotted the ear is again examined to see if the pin prick is still bleeding. The figures dealing with both these factors are recorded if they differ, but generally they do not. Microscopical examination for the formation of fibrin is not of value because it is contraction of the clot rather than its formation that we are measuring.

Conclusions.—Post-operative hæmorrhage from unsutured wounds occurs not only when an artery has not been ligatured, but also in two other groups of cases. In the first group in which the origin of the bleeding is arterial these comprise: (1) Cases in which an artery bleeds, not at operation, but afterwards on recovery from shock. (2) Cases in which an artery does not bleed at operation because the patient is too deeply under anæsthesia, but bleeds on "coming round." Chloroform mixtures rather than ether tend to produce this type of bleeding. (3) Cases in which bleeding from an artery ceases at operation because much blood has been lost, and when the volume of blood is made up by the tissues after operation bleeding may begin again. The second group, in which the origin of the bleeding is capillary, is made up of cases in which the bleeding occurs as a result of deficient clotting-power of the blood. (4) Cases may have a normal clotting-time previous to operation and suffer from a compensated acidosis following operation. The clotting-time may be prolonged and secondary hæmorrhage occur. (5) Cases in which the clotting-time is prolonged before the operation, and causes excessive hæmorrhage during the performance of it, have a tendency to secondary hæmorrhage afterwards.

In this last class there is a compensated acidosis, and if this be recognized and remedied before the operation the liability to hæmorrhage is apparently removed. In my opinion the majority of secondary hæmorrhages after operations performed by men of adequate experience are cases of this second group in which the clotting-time and not the surgeon is at fault.

In addition, I would mention that when the clotting-time is long it is very difficult to see the individual vessels which are bleeding because the whole area is oozing. This difficulty may be overcome if one puffs on a powder of bismuth or any other finely-divided powder, for then the area of capillary hæmorrhage is momentarily white, while a bleeding artery stands out a vivid red.

I wish to acknowledge the assistance of Dr. Dyke, Consulting Pathologist to the Wolverhampton General Hospital, for advice on the biochemical questions, and of Dr. Wyatt, of St. George's Hospital, for the bacteriological work.

Discussion.—Mr. J. F. O'MALLEY (Chairman) said that without any particular object in regard to hæmorrhage, he had for many years given, especially to children, some alkali three or four days before operating, and prolonged the administration for a day or two after it. It had been pointed out to him that it was valuable from the point of view of acidosis, and probably it was useful from the standpoint of hæmorrhage. At the Evelina Hospital, years ago, he had carried out a series of operations under various anæsthetics, and his conclusion was that more post-operative hæmorrhage occurred with chloroform than with ether or nitrous oxide. Crile had found the blood-pressure normal under nitrous oxide, nearly normal under ether, but depressed under chloroform, indicating in the latter case a poisoning of vasomotor control, which would account for the blood-pressure taking a longer time to return to normal; there was also a relaxation of vessels, permitting hæmorrhage.

Mr. HERBERT TILLEY asked whether the post-operative bleeding in removal of tonsils had occurred in those cases in which the main arterial supply had been ligatured during the operation, or merely controlled by pressure. It was his (the speaker's) custom to tie the tonsillar branch of the descending posterior palatine artery in every case—child or adult—and to this precaution he ascribed the fact that during the past four years secondary hæmorrhage had occurred only in three of his cases. Dr. Yates' observations would seem to be particularly valuable in cases of recurrent epistaxis.

Mr. M. VLASTO referred to the case of severe hæmorrhage mentioned by Dr. Yates, who stated that the hæmorrhage ceased only when the clotting-time returned to normal. He (Mr. Vlasto) felt that the assumption that the clotting-time was the only factor in the cessation of hæmorrhage was not justified. The fall in blood-pressure following a severe hæmorrhage would have to be taken into account. He asked Dr. Yates to explain the technique employed to ascertain the clotting-time in such cases. Whenever possible he avoided ligaturing vessels in the tonsillar fossæ after operation; there was less risk of post-operative hæmorrhage if the vessels were allowed to retract through the muscular layer than if a ligature were applied which was liable to slip off when the patient was coming round. Nor had he found any effect on the coagulability of the blood arising from the administration of calcium salts, and he was under the impression that those who used the drug as a routine were acting on tradition more than on the result of observation.

Mr. W. THACKER NEVILLE said that a good method of finding the bleeding points was to take the enucleated tonsil and plunge it into the tonsillar fossa. This arrested the general oozing and allowed one to see the bleeding points under general anæsthesia. He always ligatured, usually employing two or three ligatures, but sometimes putting six ligatures into each fossa after removing a small fibrous septic tonsil. The ligatures did not slip, as he undermined the bleeding points with a ligature on a small ⅜ curved needle. Under local anæsthesia he employed Professor Portmann's method; there was usually no bleeding point to ligature.

Mr. MACAVENER asked how soon the clotting-time could be lowered in a given patient; was sodium bicarbonate the best alkali for the purpose, and were the results all obtained by mouth administration, or was intravenous injection likely to give quicker results?

Sir JAMES DUNDAS-GRANT asked whether a distinction was to be drawn between acidosis, such as followed starvation, with diminished alkalinity of the blood and hyperacidity of the urine; also, what kind of cases were deficient in calcium, and therefore requiring the administration of alkali? The method given for testing the coagulation-time quickly was certainly valuable. For a number of years he had given calcium by the mouth, and he believed it resulted in quicker coagulation. How was the acidity of the urine most conveniently tested? Hæmorrhages, he noticed, usually occurred when the patient was surrounded by solicitous friends. He had devised a special apparatus in the form of a tube

for applying a ligature, but often the Spencer Wells forceps had come off, and the procedure proved unnecessary, as the bleeding had stopped.

Dr. YATES, in answer to Mr. Tilley, said he dissected out first one tonsil, packed the bed if necessary and then dissected out the other. He then removed the adenoids, packed the naso-pharynx with gauze, introduced a special airway and packed gauze round this into the tonsillar beds. He took the coagulation time of the blood running out of the nose. He allowed the gauze to remain for that time, and then tied the vessels, which were still bleeding. Five years ago he had had many cases of post-operative bleeding, but during the four years he had used this method he had only had one case of it.

In answer to Mr. Vlasto's question concerning the case of rheumatism, as already stated, the man weighed 14 stone; he did not know the blood-pressure, but he had a very full pulse, both before and after the operation. If a patient lost much blood, it was not failure of the circulation which stopped the hæmorrhage, but the change in the clotting-time. If one sliced a tiny vessel longitudinally or button-holed it, the person would bleed to death unless there were a means of stopping it; he considered that the means was the alteration in the clotting-time.

As to knowing in what case one should give sodium bicarbonate he knew of no certain method of recognizing this. From clinical observation it was evident that most people with a greasy skin were "bleeders." Patients who had much sepsis in the nose, pus running down the nasopharynx, could easily be tested, and many had quite a long clotting-time. The blood in the record case he had encountered took sixty-five minutes to clot, and if he had operated upon that case he would have had a very serious time of it.

The period required to get the urine under the influence of the alkali varied a good deal with the condition of the patient. If one knew that the patient had a compensated acidosis one could take time, and the results were better; a fortnight could be taken. If a quicker result were desired the sodium bicarbonate could be given per rectum. Too much alkali could be given, with resulting alkalinosis, a condition to be avoided, although it probably did not matter so much in the adult, except in a few rather rare conditions, but it was certainly dangerous in children. He had found sodium bicarbonate the best alkali to use. Magnesium hydroxide had the disadvantage of being a purge. He had not experienced any alteration in the clotting-time following the administration of hæmoplastin. Calcium given intramuscularly generally stopped hæmorrhage, but the local abscess resulting contra-indicated its use.

The Pharyngeal Pouch of the Pig.

By DOUGLAS GUTHRIE, M.D.Edin.

[ABSTRACT.]

Dr. GUTHRIE demonstrated specimens illustrating the pharyngeal pouch normally present in the pig, but which is not found in any other domestic animal. Nothing is known of its function.

The position of the pouch, in the posterior wall of the laryngo-pharynx, is precisely the same as that of the pharyngeal diverticulum which occasionally appears in man.

The pouch is briefly described in works on veterinary anatomy.

The pharyngeal pouch of man is usually regarded as a hernia which develops gradually, but the pouch in the pig is in no sense a hernia. It is a congenital structure, and contains muscle fibres in its wall. A further study of this pouch in the pig might add to our knowledge of the causation of the pharyngeal pouch encountered in laryngological practice.

Silent Tracheotomy: Its Significance.

By A. J. M. WRIGHT, F.R.C.S.

THE two incidents here related have taught me certain general principles of considerable importance. The first case I have already reported anonymously in the *British Journal of Surgery*, 1920, vol. vii, p. 549, as an "instructive mistake."

A submucous resection of the septum was performed on an adult male, the nasal passages being plugged on each side with a rubber glove finger filled with gauze. As he was being wheeled from the theatre, it was noticed that he was not breathing and was somewhat cyanosed. On commencing artificial respiration no air entered, and I then noticed that the plugs were missing from the nose. I performed tracheotomy, but on resuming artificial respiration no air entered the tracheal wound. I then passed Paterson's direct laryngeal forceps through the tracheotomy wound down to the tracheal bifurcation and extracted first one and then the other plug. After further artificial respiration, the patient recovered.

The second incident bore many points of similarity and took place in March last.

After removing tonsils from a boy of six, under chloroform and ether, with a guillotine, I proceeded to curette the nasopharynx. While I had the sensation of removing a considerable mass, nothing came out with the curette. Respiration at once ceased and was not resumed in spite of holding the child up by the feet and pressing the thorax. I therefore performed a tracheotomy and extracted a large mass of adenoids from the bifurcation of the trachea, at the first attempt, with Paterson's forceps through the tracheotomy wound. The child recovered.

A foreign body in the air passages may be impacted in the glottis, at the tracheal bifurcation, or in a bronchus. We are all now very familiar with the signs and symptoms of a foreign body in the bronchus, but the bronchial foreign body differs from the other two in that the symptoms are not of great urgency. A foreign body impacted in the glottis is easily recognized. Only seldom, however, can it happen that a foreign body of such a size and consistency as to plug the tracheal bifurcation can pass the glottis. In both of these cases this took place, and the signs and symptoms produced were so similar as to be characteristic. Cough was entirely absent, probably owing to impaction taking place at the beginning of inspiration with the lungs relatively empty. No obvious attempts at spontaneous respiration were observed, the whole picture resembling an anæsthetic overdose. The only noticeable point in which the cases differed from an anæsthetic overdose was the absence of air entry on artificial respiration. In the first case, the disappearance of the nasal plugs gave a clue, while, in the second, the sensation of curetting through the mass of adenoids without their production, combined with my previous experience, supplied the diagnosis.

On opening the trachea, the absence of all sound was extremely striking, and I was surprised at the difficulty in ascertaining when the trachea had been entered owing to this absence, particularly in the case of the child, in whom the rings were naturally soft. Once the trachea has been opened, forceps passed down through the wound will, in such a case, inevitably seize the foreign body, and there is, therefore, no need for, neither is there indeed any time for, the use of direct vision.

That every effort should be made by the surgeon to prevent the inhalation of foreign bodies is so obvious that it needs no further emphasis. I wish, however, to draw particular attention to the following conclusions:—

(1) Soft foreign bodies may sometimes pass through the glottis and entirely obstruct the tracheal bifurcation. This accident does not necessarily produce obvious signs of asphyxia; therefore, if the patient is under a general anæsthetic, a mistaken diagnosis of overdose may be made. I think it probable that some deaths during the removal

of tonsils and adenoids are really of this nature. (2) The absence of air entry, on artificial respiration, indicates an obstruction in the air passages; a similar absence, after tracheotomy, indicates that this obstruction is at the tracheal bifurcation. Such a foreign body can be rapidly and certainly removed by the blind introduction of forceps through a tracheotomy wound. We now always have such forceps put out with the tracheotomy set when operations on the nose and throat are being performed.

Discussion.—Mr. MUSGRAVE WOODMAN related an accident which had recently happened in his practice. He was removing tonsils by dissection, and at the conclusion of the removal of the first tonsil he was using a crushing tool when the tonsil slipped into the back of the pharynx and disappeared. He thought it had passed to the lung. He watched for a change in the patient, but there was none. He therefore removed the other tonsil, and meanwhile sent direct for his instruments. He found the tonsil at the bottom of the right bronchus, and brought it up again. The tonsil had passed beyond the danger-point, and the patient knew nothing about it.

Mr. M. VLASTO thought that the liability to accidental aspiration of blood and lymphoid tissue during operation was greatly increased by the patient being seated upright in a chair.

Mr. A. J. M. WRIGHT (in reply) said he no longer used an untethered finger-stall. Mr. Woodman's remark was of great interest, and it demonstrated the difference between a bronchial foreign body and one at the bifurcation. The former was not a matter of life urgency, seeing that time could be taken to send for instruments.

Familial Infection of Chronic Sinusitis: its Clinical Import.

[ABSTRACT.]

By PATRICK WATSON-WILLIAMS, M.D.

Dr. P. WATSON-WILLIAMS said that although it seemed inevitable that chronic as well as acute rhinitis should be contagious if only in a less degree, yet this might not be easy to prove. When confronted with fibrinous rhinitis, due to a distinct organism—the Klebs-Loeffler bacillus—contagion is readily proved; but, so ubiquitous is the common cold, that we take refuge in the belief that while certain families tend to escape, others are predisposed to catarrhal infections. In a measure this is doubtless true; nevertheless there is a difference between proclivity to common colds which come and go completely, and a chronic catarrh when it becomes an "established" or "focal" infection.

Now if, as there is good reason to believe, chronic nasal focal sepsis is a causal factor in appendicitis, gastro-intestinal catarrh, etc., we ought to be able to find that such abdominal sub-infections prevail in families with a proclivity to chronic naso-oral sepsis. Of course, similar evidences of a tendency to sub-infections in other territories should appear, but we shall restrict our investigations to the abdominal region, except for one example in which mastoiditis and insanity seem to have resulted from familial infection.

He (the author) suggested that a child, subject to a nasal sinus focal infection, rarely escapes septic infection of its tonsils and adenoids, and that when these alone have been removed, the persistent re-infection of the remaining lymphoid tissue causes recurrence of adenoids. But this child will tend to infect brothers and sisters, though auto-immunization may in time confer a relative immunity on the offender. But if that child grows up and marries, this parent is prone to infect members of his family. In some cases his investigation for such unsuspected sources of infection in children has shown that a parent has been the subject of a long-standing nasal sinus infection.

What has been accepted as inborn familial constitutional weakness might be more correctly explained as "carrier infectivity." Many examples of such family histories were shown in tabular form; one of many examples had previously been recorded in the *Journal of Laryngology, Rhinology and Otology*, vol. xxv, April, 1920.

He quoted another example from his Semon Lecture (1925), illustrating "The infectivity of chronic nasal sepsis and, in addition, its causal relationship to appendicitis where a parent may act as a carrier infecting the children."

(*a*) Father, aged 58. Catarrhal otitis for thirty years. Chronic sinusitis (operation fifteen years ago and still infective three years ago). He had five sons (no daughters); of these four had definite nasal sinusitis and only one had escaped operation for appendicectomy.

After exhibiting a number of other examples on the screen, not hitherto recorded, he concluded by stating that there was good reason to believe that early and effective treatment of parental nasal infection would have saved many serious abdominal operations alone, without taking into consideration conveyed infection in other territories.

Discussion.—Mr. W. STUART-LOW said that he was called in consultation to the son of a farmer in Oxfordshire, who was suffering from an acute frontal sinusitis. He found the farmyard full of mud and slime; the surroundings were very insanitary and the atmosphere misty and damp. On his advice the family moved to a sunny slope in the south, and thereafter enjoyed good health. "Surroundings" had much to do with the origin of such conditions as those described by Dr. Watson-Williams.

Mr. MUSGRAVE WOODMAN said that many cases of chronic nasal catarrh in young children seemed to resist cure. They were cases in which tonsils and adenoids had been removed. There was not yet much evidence of gross sinusitis, yet nasal catarrh was present. His attention was directed to this group by a striking example which occurred three years ago. He was asked to treat quinsy in an infant of nine months, and he wondered how it had become infected with that condition within a year. The father had had suppurating sinusitis, pus streaming from the sphenoid, and the mother had had five attacks of quinsy. It was clearly an instance of direct infection from the mother. In cases of that kind he went into the family history as well as that of the patient, also he asked to see the nurse. Often the nurse was the centre of infection, and in many cases she was more in contact with the child than anyone else.

Dr. LOWNDES YATES said that he once had a group of cases in which children had had a definite discharge from the nose which did not yield to treatment. It was observed that the mothers of many of these children were suffering from pyorrhœa, and that when the sepsis in the mothers' mouths had been removed the discharge from the noses of their children disappeared. This raised the question as to whether the infection could be air-borne. Much work had been done on this question in France, but no proof was forthcoming that the infection was air-borne. It might be conveyed by fingers inserted in the nostrils. Still, in the case of children, there was good evidence that the infection might be conveyed by the mother or a nurse if they had infected teeth or well-marked sinusitis.

Dr. J. S. FRASER agreed that environment was important; but there was also the family diathesis, as it was formerly termed. The late Sir Thomas Grainger Stewart laid great stress on diathesis, and it might be there was a catarrhal diathesis. It was recognized that there were families in which the chronic nasal discharge was very common. Everybody at some time or another suffered from acute nasal catarrh, but fortunately not in all cases did it become chronic. Might it not be that there was some inherent weakness in these families which tended to be manifested in the development of chronic nasal catarrh? This condition might be due to susceptibility rather than to direct infection from a relation.

Mr. JAMES ADAM said he thought that deficiency of vitamins and excess of carbohydrate would cause such conditions. In the hospital to which he (the speaker) was attached there were 400 beds for children, and when he first went there he was continually troubled by running noses and sinusitis in these children. He began open-air treatment, dosage with a little cod-liver oil and the eating of an apple daily; also handkerchief drill; since then there had been practically none of these chronic cases.

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Mr. H. S. BARWELL (President) said it seemed that there were various possibilities to be considered in familial catarrhs, or infective conditions: direct infection, environment, the possibility of vitamin deficiency in the diet and endocrine disturbance; and lastly, diathesis (which might be regarded rather as a diagnosis to be arrived at after exclusion of the other three).

Dr. WATSON-WILLIAMS (in reply) said he only wanted to elicit interest in this question on very broad principles. With regard to the remark of Dr. Fraser on familial diathesis, the fact remained that, in spite of any predisposing diathesis, many affected members of families were cured of trouble when the sinuses and the upper respiratory tract had been rendered healthy: diathesis did not then seem to operate. The report of these cases, again, emphasized the desirability of removing all sources of established sepsis. As to bad environment, it did probably act as an influence at times; but the cases reported were all cases of patients from families in good circumstances residing in various districts and all considered particularly healthy, so that it could not have been a contributory cause in all these cases.

Intrinsic Cancer of the Larynx operated through a Laryngo-fissure. (Lantern Demonstration of Immediate and Lasting Results in Seventy Cases.)

By Sir STCLAIR THOMSON, F.R.C.S.

[ABSTRACT.]

THE author said he had recently completed a series of seventy cases of laryngo-fissure for intrinsic cancer of the larynx, apart from those in which he had followed this route in order to treat enchondroma or for exploration. He had obtained the after-history of each of the seventy cases. This series extended over twenty-eight years, as the disease, compared with malignant disease elsewhere, such as of the breast, was very rare. His object in these cases, now, was to go as far down into the subglottic space as possible and up on to the ventricular band, and to remove everything between those points, including the internal perichondrium, leaving only the external perichondrium. The dangerous area was the subglottic space, and he liked to take away not only the anterior commissure, but a little tissue to the other side of it.

Laryngo-fissure gave an excellent result. Sixty cases were private and ten were hospital patients. In females the disease occurred earlier in life than in men, but in males its onset continued later. Of the seventy patients, thirty-four were still alive, three to nineteen years after operation. There had been eighteen deaths from causes other than recurrence and three deaths from operation—namely, one from rupture of the œsophagus due to vomiting, and two from bleeding; these two patients would probably not have died if they had not been given heroin or morphia. He never used "dope" drugs either before or after operation. Eleven of the patients had developed malignant disease elsewhere, including glands in the neck. Most of the recurrences were within the first year, this showing the importance of Semon's instructions to patients to present themselves once every month for the first year. In borderline cases, should there be recurrence, laryngectomy should be performed. In subglottic cases—those in which the cord was fixed—the prognosis was unfavourable. The author considered that there should be no operative death in these cases unless something exceptional occurred. The operation left the patient with an adequate voice and no social disability, and in selected cases the cure should be lasting. He had in the past operated upon cases such as, with a larger experience, he would not attempt now. Of nine cases in which the cord was fixed, only four patients lived more

than three years after operation. He alluded to the importance of a careful preparation of the patient. He was not insistent upon any particular form of anæsthesia; the operation could be done under local anæsthesia, and if a general anæsthetic was preferred the patient should not be under it very deeply. No atropine, morphia, or similar drugs should on any account be used, but bromide was permissible, or aspirin. He did not see any advantage in using cocaine beforehand, nor in plugging the larynx, nor in doing endo-resections. There was still need to urge that these cases should be sent to the laryngologist at an earlier stage, for that was, naturally, the stage at which operation gave the best results.

Discussion.—Mr. H. S. BARWELL (President) said that the more the record of such experiences could be heard the better, because these cases varied so much that the more instances one could hear carefully described and see illustrated, the better would be the chance of laryngologists dealing successfully with such cases which came their way.

He also had been impressed with the rarity of such cases in hospital practice (i.e., cases at a stage suitable for laryngo-fissure) in comparison with those seen in private practice. Sir StClair had eight in private to one in hospital. Sir Felix Semon told him (the speaker) years ago that he had never seen in hospital a case suitable for laryngo-fissure, but at the present day patients with the disease presented themselves at a somewhat earlier stage. The family doctor needed to be more educated as to the sending of patients with hoarseness of more than a few weeks' duration, which did not quickly improve under treatment, to an expert in order to have the larynx examined. And the public also needed to be told that the throat should be thoroughly examined in all cases of persistent hoarseness.

Professor GEORGES PORTMANN (Bordeaux) said that since he had seen Sir StClair Thomson perform laryngo-fissure he had become a warm supporter of the operation. In the tables shown were cases of patients who had lived 11 years and 12 years after removal of glands, and he would like to know what Sir StClair's practice was in that respect; did he take the glands away at a second sitting or remove them months or years after the primary operation?

Dr. F. R. PACKARD (Philadelphia, U.S.A.) said that he had had very little personal experience in this class of case. He and his colleagues frequently wondered that the term laryngo-fissure was not replaced by the name partial laryngectomy, or hemi-laryngectomy. In America it was felt that "laryngo-fissure" simply implied an incision in the larynx, and not a dissection of the growth.

Mr. W. STEWART asked whether, in a case in which Sir StClair Thomson found the cord fixed, he did laryngo-fissure, or did he go straight on to perform laryngectomy?

Mr. THACKER NEVILLE said he was a beginner in this operation, as he never saw cancer of the larynx in China. In the last year he had carried out three laryngo-fissures. The first patient died from hæmorrhage, which began when in operating he got to the arytenoid. He tried ligaturing and then applied a cautery, and the hæmorrhage stopped. The patient did well, and was to be discharged from hospital on the eleventh day after operation, when on the tenth day he had a severe hæmorrhage and died. His mistake had been employing the cautery instead of ligaturing. In the second case the anterior two-thirds of the left vocal cord was involved. At operation he found the growth had crossed the middle line and so he cut through it in making the fissure. He removed the left vocal cord and anterior one-third of the right. He did not remove cartilage and there was a recurrence for which the patient refused further operation. His third case was one of papilloma of the vocal cord, which was easy to remove. He ligatured the vessel at the arytenoid by under-running it with a ligature on a needle and, as recommended by Sir StClair, he removed the cartilage and the patient did well. The report of the pathologist was that it was tubercle.

Dr. J. B. CAVENAGH said the public could not be expected to have knowledge of such subjects; improvement in regard to seeing cases earlier must depend on the doctor. At the present day the subject was doubtless taught in the medical schools. But it also devolved upon doctors to bring the matter before their own local medical societies; this was rarely done, hence the opportunities of obtaining cases early for treatment were largely lost.

Dr. J. S. FRASER said he had operated upon twenty cases of intrinsic cancer of the larynx, ten of them hospital patients. One was sent with a history of hoarseness of only one month.

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He had been surprised to see how much of the false cord Sir StClair removed. He took it that the malignant disease spread downwards to the subglottic region, rather than upwards to the false cord. In one case in which he (the speaker) had removed a good deal of false cord there was some difficulty in swallowing afterwards, and for some days the patient had to swallow with his head over the edge of the bed, and to take food by sucking through a tube. He recently saw a case in a patient from New Zealand in which there had been hoarseness three and a half years, and yet the affected cord was very mobile. It had a "mouse-eaten" appearance, but towards the vocal process there was a papillomatous growth, which he removed. The report was "squamous epithelioma." He saw the section, and the patient was subjected to operation.

With regard to cancer occurring elsewhere, the only case he had had of this kind was in a woman whose larynx was excised, and she died seven years later of cancer of the cervix uteri.

He emphasized the importance of a thorough preliminary general medical examination of these patients. In the case of at least two of the patients he had operated upon, in which death took place within a week of operation, the medical condition was such that the operation ought not to have been done. These cases had been medically examined but not with sufficient care.

Sir StClair Thomson (in reply) said he was pleased to have elicited the questions he had been asked, as they were all of interest. Sir Felix Semon once told him he had not seen a case in hospital suitable for operation by laryngo-fissure, and that showed what good work could be done in private practice.

In one of the cases in which recurrence took place in the glands there was no recurrence locally. He handed the patient over to Mr. Trotter, as a very deep operation was necessary. That was about eight years ago, and recently he had seen the patient, quite free from disease in larynx and in neck.

He was glad to hear Dr. Packard insist that this operation was really a partial laryngectomy. This was an instance of how we were sometimes the victims of words. By saying it was enough to "split the larynx and take out a cord," people were misled as to the extent of the procedure necessary. In all cases of malignant disease, in any part of the body, one should try to get a face view of it. That was why he was opposed to window resection. He removed the diseased area in as wide an extent as possible, short of crippling the patient. That was why the ventricular band was removed in the case to which Dr. Fraser alluded. The pathologist often reported that the growth had spread along the cord towards the floor of the sinus of Morgagni; sometimes he felt doubtful whether he had removed all the cancer from the muscle layer. If the operator entered deeply, he had to take away the ventricular band. The subglottic region was certainly dangerous, and he had been surprised that even with a mobile cord the disease might yet be deep in the muscles.

He had been asked whether he operated when the cord was fixed. He hoped to publish later, not from a mere impression, but from a careful study of his case-papers, a definition as to what cases were suitable for the operation. But he could try to outline them now.

If a growth was limited to a mobile cord and it had not extended to the anterior commissure, or the arytenoid, it was a most suitable case for treatment by laryngo-fissure. If the growth had extended to the anterior commissure it was operable, but not so hopeful as to result as in cases of the previous kind. If the growth had extended to the vocal process, close to the arytenoid, he would not operate. If the growth had invaded the subglottic space and the mobility of the cord was impaired, it was a doubtful case. If the cord was absolutely fixed, it was very doubtful, though he had had successes even in that class of case. In these borderline cases the decision must rest with doctor and patient. If it were the case of a young patient who had to earn his living, one would give him the benefit of laryngo-fissure, watching him once a month and preparing to do laryngectomy if there were a recurrence. If the patient had retired from his occupation and was in good circumstances and desired to be freed from all anxiety, he (the speaker) would do complete laryngectomy. But sometimes, as others had observed too, one cord would be affected and the other appear to be free, but yet the growth had penetrated into the subglottic area and had crossed to the subglottic area on the other side by way of the anterior commissure. The patient should be thoroughly cocainized, and inspected several times.

It was praiseworthy of Mr. Thacker Neville to have related his misfortunes in early cases. The death from hæmorrhage in that gentleman's patient was a surgical misfortune; both Sir Henry Butlin and Sir Felix Semon used the cautery in early days, but because it conduced to secondary hæmorrhage they gave it up.

Dr. Fraser's criticism as to a third of his cases being in hospital patients was interesting. A thorough medical examination was admittedly important, but there again came in the "glorious uncertainty of life." One of his (Sir StClair's) patients was aged 59; he had him medically examined. The physician warned the speaker that the blood-pressure was 200, and the arteries hard. Rather than let him die miserably, however, operation was decided on; there was no hæmorrhage, nor cause for anxiety, and he died aged 72, of cerebral hæmorrhage without any recurrence.

LARYNGOLOGICAL SESSION, JUNE 1, 1928.

CLINICAL MEETING.

Chairman—Mr. HAROLD BARWELL, F.R.C.S. (President of the Section of Laryngology).

Laryngeal Case for Diagnosis.

Further Report of Case previously shown.[1]

By M. VLASTO, F.R.C.S.

MANY Members of the Section who saw this man, aged 31, who had suffered from hoarseness for about a year before being shown, considered that it was a case of laryngeal tuberculosis. A piece of the growth on the right cord had been removed for examination, and on Dr. Elworthy's report a laryngo-fissure was performed in June.

Sections of the growth are shown.

Discussion.—Professor G. PORTMANN said he had seen this patient, and saw no proof in the section that the condition was malignant, though the state of some parts of it seemed doubtful. It was very important, from the points of view of diagnosis and prognosis, to see other sections.

Dr. JOBSON HORNE suggested that the case be referred to the Morbid Growths and Research Committee for report. The report, with a specimen of the growth for the Society's Cabinet, would give the completeness to the case required for scientific and statistical purposes.

Mr. VLASTO replied that he did not feel competent to express an opinion on the nature of the lesion. Dr. Elworthy reported from the small piece taken away that the growth was definitely malignant. In spite of several opinions that it was tuberculous, he (the speaker) had carried out a laryngo-fissure. He was anxious there should be no mistake, and he would send the section to the Morbid Growths and Research Committee for report.

[1] See *Proceedings*, 1927, xx (Sect. Laryng.), p. 46.

98 Davis: *Hyperostosis Cranii; Osteoma of the Orbit and Maxilla*

Hyperostosis of the Maxilla.

By E. D. D. DAVIS, F.R.C.S.

PATIENT, a female, aged 57; has had a tumour of the left upper alveolus for more than twenty years. She was first seen in July, 1917, and the swelling has slightly increased.

X-ray photographs of the skull and other signs indicate osteitis deformans.

Hyperostosis Cranii.

By E. D. D. DAVIS, F.R.C.S.

W. T., MALE, aged 38, was shown to this Section in January, 1912. He then had symmetrical osseous swellings of the nasal processes of the maxillæ extending on to facial surfaces of the bodies of the maxillæ. Both sides of the nose are obstructed by the hard osseous swellings which can both be felt and seen projecting into the nasal cavities. The infra-orbital margins are involved and there is lachrymal obstruction, but in other respects the orbits are apparently normal.

In addition there is a diffuse, smooth swelling of the body of the mandible to the right of the mental eminence and surrounding the mental foramen. The bony growths have slightly increased during the last sixteen years.

X-ray photographs are shown with the patient. Both maxillæ are occupied by dense masses of bone.

Osteoma of the Orbit and Maxilla.

By E. D. D. DAVIS, F.R.C.S.

PATIENT, a female, aged 65, first seen in October, 1924. She has a large osteoma of the maxilla which displaces the left eyeball forwards and outwards. The left eye is almost blind. The left nasal cavity is obstructed by the growth. Wassermann reaction strongly positive. The lower portion of the osteoma in the canine fossa is necrosed, and a piece of it, about the size of an egg, was removed twelve months ago. At the same time the growth was explored from this site and it was then decided, owing to sepsis, that a complete removal of the osteoma was impracticable.

Discussion.—Dr. SALISBURY SHARPE said the appearance in the second case of hyperostosis was absolutely identical with the tropical disease "goundou," a condition of hyperostosis in the nasal processes and nasal bones. The ætiology of it had only recently been fully worked out. It was now regarded as a sequel of yaws, a spirochætal disease allied to syphilis. The Members of the Royal Society of Tropical Medicine and Hygiene and of the Tropical Diseases Section would probably be glad to see the case.

Mr. E. D. D. DAVIS (in reply) said the first of the cases was typical; there was a localized hyperostosis of the palate and alveolus. Years ago, Sir Victor Horsley operated upon five

similar cases; he found a very thick and vascular periosteum, and beneath it spongy new bone. Sir Watson Cheyne operated upon a similar case, whom he (the speaker) watched for some years.

In the second case of hyperostosis the Wassermann was positive, and the man had been treated for syphilis, but it made no difference, hence it was concluded that the positive Wassermann was accidental. No further investigations had thrown additional light on the case.

The Wassermann reaction was positive also in the case of osteoma of the orbit and maxilla, and the patient had necrosis of a portion of the osteoma in the cheek sulcus. The odour before a large sequestrum was removed was overpowering.

Lymphangioma of Larynx. (Tumour and Section shown.)

By A. R. TWEEDIE, F.R.C.S.

JUNE 15, 1927.—Male, aged 57, complained of nasal "obstruction" due to fall on nose during infancy.

Examination.—Nose: gross anterior obstructive irregularity of nasal septum, chronic naso-pharyngitis. Pharynx and post-nasal space otherwise normal. Rounded red neoplasm over posterior half of left ventricular band discovered in course of routine examination, at which nothing else abnormal was found.

June 25, 1927.—Under local anæsthetic, using Haslinger's directoscope. Tumour semi-sessile, easily grasped with forceps and removed with knife. Site of origin was junction of posterior end of left aryteno-epiglottic fold and ventricular band. Apart from some slight consequent laryngitis, normal convalescence.

August 10, 1927.—Site of tumour was now represented by small subsiding infiltrated area. Patient not seen since.

Dr. J. Kilian Clarke (Pathologist to General Hospital, Nottingham) reports as follows:—

"The tumour is composed of round cells set in a fibrous tissue stroma, the whole being covered with stratified squamous epithelium. In places there is a tendency to alveolar arrangement, while in others lumen formation can be seen. The growth is, in my opinion, endotheliomatous in nature, and is probably an angioma."

As such tumour is very rare in the larynx, I sent a section to Dr. W. D. Newcomb, Pathologist to St. Mary's Hospital, Paddington.

He replies: "I believe it is a lymphangioma of rather cellular type. It is rather like one we had some years ago under the skin of the back. Provided it was all removed, it should not recur. I have never seen anything like it before in the larynx."

Discussion.—Mr. A. R. TWEEDIE said that the case was shown in relation to a similar case recently brought forward by Mr. Horsford. He would be very pleased to present the specimen to the Museum.

Mr. CYRIL HORSFORD said he had since removed the growth in the case referred to, and it looked like angeioma. The microscopical report was that the growth contained epitheliomatous tissue.

100 Dundas-Grant—Jobson

Tumour Growing from the Lower Part of the Left Tonsil and Base of the Tongue.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT a male, aged 63. On inspection, the oropharynx was seen to be almost completely filled by a rounded tumour, smooth on its anterior surface, but rugged on the posterior. It appeared to grow from the lower part of the left tonsil and base of the tongue. At first sight it looked inoperable, but on careful palpation under an anæsthetic its continuation into the outer wall of the pharynx seemed rather of the nature of a burrowing than of an infiltration. This was confirmed by Mr. Percival Cole. After incision of the mucous membrane we were able to shell out the growth from its surroundings, and to remove it by morcellement with apparent completeness. Mr. Cole stitched a tube of radium into the opening left in the side wall. The patient now swallows and breathes quite easily, though before the operation swallowing was almost impossible and breathing difficult.

Dr. Fry reports as follows .—

"Sections show an alveolar arrangement of closely apposed polyhedral or spheroidal cells closely applied to the outer surface of capillaries. The latter contained well-formed red cells in many instances, and the endothelial cells lining them are often prominent and hyperplastic. The nuclei of the cells are large, and stain well. There is very little intercellular connective tissue, the supporting tissue being largely composed of small vessels or capillaries. There are areas of hæmorrhage amongst the cells. In general, the sections suggest a perithelioma derived from the blood-vessels. Alternatively, it bears considerable resemblance to a carotid body tumour, of which it might conceivably be an extension. These are often regarded as peritheliomas. Finally, it is conceivable that it might be a secondary nodule from a spheroidal-celled growth, possibly arising in the thyroid."

Discussion.—Mr. E. D. D. DAVIS asked whether the tumour was encapsuled. The appearance suggested a mixed tumour; such growths did occur in that situation.

Sir JAMES DUNDAS-GRANT replied that the tumour was fixed and not encapsuled at its site of origin. There was some resemblance to a carotid body tumour, but further comparison showed that it was not identical with the latter.

Anti-Mouth-breathing Mask.

By T. B. JOBSON, M.D.

THIS is a device for keeping the mouth closed after operations for producing an adequate airway.

A wax impression is taken of the lower half of the patient's face. From this a cast is made. From the cast a thin metal mask is modelled. This fits accurately the lower half of patient's face from the lower margin of the anterior nares to below the chin. Suitable holes are drilled in the metal. These enable a lint lining to be tacked on and two thin elastic bands attached. The mask is held in position by these elastic bands, which are fixed to a head band of stout webbing by means of "hooks and eyes." The mask has been used by exhibitor for the past six years and found very efficient. The exhibitor desires to thank Mr. C. H. Housden, L.D.S., for his help in making the mask.

This mask is easily made by any dentist, but it is better to have one prepared for each individual patient, in order to ensure a better fit. It exerts pressure on the upper lip, which has a good effect in regulating the pressure on the teeth in the upper jaw. It is efficient, keeps its position, and is comfortable.

Laryngeal Case for Diagnosis.

By T. B. JOBSON, M.D.

MRS. B., aged 58. Husky voice for a year or more. "Tight" feeling in throat for twelve months. Lately she has had alarming attacks of spasmodic dyspnœa. States that she has not had scarlet fever or diphtheria, but had an "ulcerated throat" seven years ago. Wassermann negative. Soft palate shows scarring. The laryngeal image is extraordinary—the larynx being covered over with a pinkish growth, in the centre of which is a small elliptical opening through which the cords are visible. There is no complaint of dysphagia.

Discussion.—Dr. JOBSON HORNE considered that the condition on the soft palate and in the larynx was scarring and cicatricial tissue resulting from lupus or syphilis, or both.

Sir JAMES DUNDAS-GRANT said he regarded the condition as lupus; he had several times seen a similar appearance presented by that disease.

Mr. HAROLD BARWELL (President) said this seemed to be entirely cicatricial, with narrowing at the upper aperture of the larynx, and with destruction of soft palate, uvula, and epiglottis. The lesions must have been present much longer than the patient realized.

Laryngeal Case for Diagnosis.

By T. B. JOBSON, M.D.

MRS. S., aged 55. History of hæmoptysis three months ago, and a second attack a month later.

Cough present but no sputum. Voice a little husky.

On the posterior surface of the epiglottis there is to be seen a bright red tumour about the size of a small cherry. The posterior half of the tumour presents a dirty yellow membranous appearance. A swab from this surface yielded "staphylococci only."

Discussion.—Mr. BELL TAWSE suggested it was of angeiomatous nature, and should be destroyed by diathermy.

Dr. JOBSON replied that a month ago the tumour was four times the present size, and completely blocked a view of the cords. The case was sent up as one of phthisis, and the doctor who sent it said there was a patch of disease in the lung, and asked for a report on the larynx, because the voice was husky. The cause of the hæmoptysis was found to be in the larynx, which was unusual; an unexplained hæmoptysis was usually due to a lung condition. He regarded it as angioma, and it was his intention to treat it by diathermy. He had postponed treatment so that Members might see it, Meantime the patient had been curing herself, each hæmorrhage apparently reducing the tumour still further.

Mr. THACKER NEVILLE said that the diathermy snare could be used.

102 Tilley: *Frontal Sinus Associated with Severe Headaches*

A Very Small Frontal Sinus associated with Severe Headaches. No Suppuration Present.

By HERBERT TILLEY, F.R.C.S.

MISS R., aged 29.

History.—Severe headaches over left eye, every four to five weeks, for ten years, which began in the morning and reached their climax about 6 to 7 p.m. The most acute pain was felt in the region of the outer angle of the palpebral fissure, but a more generalized pain extended from the supra-orbital region to the vertex. Patient had to keep to her bed during an attack.

Examination.—No external abnormality. Pressure over and beneath the left frontal sinus caused a dull pain. No discharge from left nasal cavity, which appeared to be quite normal.

Radiogram suggested complete absence of sinus.

Treatment.—Mr. Trotter, in whose care the patient was, agreed that it would be wise to explore the condition of the bone in the region of the sinus. I did this on March 6. The antero-inferior wall of the frontal bone corresponding to that of a normal sinus was between $\frac{1}{8}$ and $\frac{1}{2}$ in. thick and very vascular. Only a very small cavity was found at the upper end of the fronto-nasal canal. The wound was sutured and healed by primary union. The headaches have been greatly relieved.

Discussion.—Mr. BELL TAWSE asked whether the increased vascularity of the bone was the cause of the headaches, or was the cure of the headache a psychological effect of the operation?

Mr. HAROLD BARWELL (President) said he was sceptical about the cure of the headache as a direct result of operative interference. Every Member probably had had cases of pains about the head, in which the patients had been cured by all kinds of operation, sometimes for a few weeks, sometimes for longer. He had seen the same in mastoidodynia; an exploratory operation was done, and the pain was completely cured, although nothing abnormal was discovered.

Mr. TILLEY replied that he shared the President's view, but the headache was so severe that the patient became incapable of continuing her work and was sent to Mr. Trotter for an opinion as to whether there was any intracerebral lesion. It was thought worth while to explore the frontal sinus as there might be some condition present which was not revealed by the skiagram, e.g., an exostosis. The bone was $\frac{1}{2}$ in. thick, strikingly congested, and bleeding from it was very free. Both the frontal and sphenoidal sinuses on that side were very small. The patient had recently written: "I have lost my headaches, and never felt so well in my life."

If anything fresh should occur in regard to the local conditions he would report again.

Removal of Orange Pip from the Bronchus.

By V. E. NEGUS, F.R.C.S.

A CHILD, aged 4, from whose bronchus an orange pip was removed four weeks after inhalation; $\frac{1}{10}$ gr. morphia given, but no other anæsthetic used.

Discussion.—Mr. W. M. MOLLISON said he did not give an anæsthetic for these cases, not even morphia, though children were given chloral. He had had a case of a child aged 14 months who had a piece of cork in a bronchus and the child went to sleep in the middle of the operation for removal.

Mr. R. P. MATHERS said it was remarkable that the foreign body was in the bronchus for four weeks; the presence of such foreign body usually caused severe pneumonia in a few days and death followed.

Mr. NEGUS (in reply) said he brought the patient because the foreign body had been there a month, and when he saw the child there was acute bronchitis, with the lungs full of pus, a temperature of 105° F. and respiration 50. As the child was nearly dead it was naturally quiet. He would not have given an anæsthetic in any case, as it would probably have led to a fatality, and when anæsthetic vapour was about it was more difficult to see with the bronchoscope. He used Jackson's bronchoscope, 4 mm.; the 5 mm. would not pass through without using force, as it was a small larynx. He showed the tube and the forceps used; if the latter were small enough it was fairly easy to see. The patient did not struggle, but seemed happier after the bronchoscope was in position. There seemed no reason why a bronchoscope should hurt a child, although passing the tube through the larynx might be painful.

Reconstruction of the Upper End of the Œsophagus.

By V. E. NEGUS, F.R.C.S.

PATIENT, a woman showing the results of reconstruction of the upper end of the œsophagus by skin flaps, after removal of a carcinoma of the pharynx. (Case previously shown in April, 1927).[1]

Discussion.—Dr. W. S. SYME said he had carried out several reconstructions; the most successful was one in which he removed both larynx and pharynx and made up the pharynx afterwards. Such work must not be undertaken unless the operator was prepared to spend a good deal of time on it, as a flap might be destroyed and one had to begin again, being content with a little closure at a time. In this case the end-result was good.

Mr. NEGUS (in reply) said that the case was brought again in order to show that the patient could still swallow comfortably. The reconstruction took a long time; he had to use two flaps because of the position of the growth; in trying to close the skin-tube the flaps once or twice failed to unite, though they did so eventually. He wished to ensure proper union so as to prevent stenosis afterwards.

Carcinoma of Right Ethmoid.

By NORMAN PATTERSON, F.R.C.S.

PATIENT, a female, aged 44. Five months' right-sided nasal obstruction with slight discharge. The right inferior turbinate presented a suspicious appearance, not characteristic, however, of malignant disease. A portion was removed on two occasions and each time the pathologist reported the presence of squamous-celled carcinoma.

On November 2, 1927, a lateral rhinotomy was performed. The disease was found to have invaded the inferior turbinate and ethmoid region. The diseased area was removed as completely as possible and diathermy applied to the cavity. At a later date, a 50 mgm. radium-container was inserted.

[1] See *Proceedings*, 1927, xx (Sect. Laryng), 62.

104 Gibb: *Spindle-celled Sarcoma of Septum Nasi*

Spindle-celled Sarcoma of Septum Nasi.

By J. A. GIBB, M.D.

F. W. C., AGED 61, first seen August 2, 1926. Complained of stuffiness in nose.

Examination.—Growth occupying left side of septum nasi—a position easy for removal.

Operation: September 12, 1926.—Perichondrium elevated from right side of septum. Cartilage and its covering perichondrium completely removed together with growth. Report: Spindle-celled sarcoma.

April 6, 1927.—Recurrence noticed on remaining portion of cartilage forming bridge, and adjoining part of ala on left side.

Second Operation: June 26, 1927.—Incision from left inner canthus, into anterior nares. Flap turned over and bridge of nose exposed. Portion of nasal bones and ascending process of maxilla removed. Growth completely removed. As there was some doubt as to the ethmoid being affected it was partly removed.

The cosmetic effect is marred by pre-existing acne vulgaris.

Sir StClair Thomson saw this case and suggested that the incision should be begun high up, so as to get a good exposure.

December, 1927.—Recurrence about size of hazel-nut seen on membranous septum.

Operation: January 21, 1928.—Incision round base of columella into nares, freed from anterior nasal spine, turned up and growth removed.

Although it is too early to forecast the result, there does not seem to be any sign of recurrence. This case is shown as an example of the difficulty of eradicating these growths by surgical measures alone.

Discussion.—Mr. W. M. MOLLISON asked whether Mr. Gibb proposed to use radium in this case.

Mr. GIBB (in reply) asked whether Mr. Mollison recommended him to use radium now, or to wait for a recurrence. [Mr. MOLLISON: Now.] The trouble was to tell these people that another operation was needed, and after that perhaps a third. Three years ago he showed two cases of the kind, a sarcoma growing from the same position as in this man's case. In these cases there was no recurrence for three or four years, but this patient had a recurrence in four months; there had been no contiguity between the growth and the area of recurrence. In one case he did two subsequent operations, and then said a third was necessary, but this was declined, Some months later the whole face had become a huge sarcoma. He would adopt Mr. Mollison's suggestion.

Extrinsic Carcinoma of Larynx treated with Diathermy and X-rays.

By G. BROUGHTON BARNES, F.R.C.S.Ed.

W. V., MALE, aged 56. Farm labourer; first seen September 10, 1927. The whole of the lower pharynx was occupied by a rounded mass covered with sloughs. It was impossible to determine its exact attachments further than that it came from the right side of the pharynx. He was breathing through a very narrow chink against the left lateral wall, with stridor. He had taken nothing but fluid, and not sufficient of that, for three weeks, and was extremely emaciated. No glands were palpable. His teeth were filthy. A piece of the growth was removed for section (exhibited), which shows masses of degenerating epithelial cells with well-formed blood-vessels. He was admitted for removal of teeth and palliative operation.

Operation (September 30, 1927).—Intended only as a palliative, to avoid permanent tracheotomy and gastrostomy. The mass of growth was removed with a diathermy

snare. The base was found to involve the right pyriform fossa, the lateral wall of the pharynx, the ary-epiglottic fold, the right free edge of the epiglottis; and it extended over the arytenoid, and down on to the back of the cricoid. Diathermy was applied to the base by means of an ordinary flat terminal and the sloughs curetted away.

He immediately began to breathe quietly and eat well, and to put on weight at the rate of about 2 lb. a week.

On January 2, 1928, the whole area treated by diathermy, the pyriform fossa and epiglottis were covered with a smooth scar, except that the ary-epiglottic fold showed an oval mass of growth. He was then treated at St. Bartholomew's Hospital by deep applications of X-ray. Six weeks later there was no growth at all visible.

The epiglottis has been gradually pulled down over he larynx by the scar. On April 18 his weight was 28½ lb. more than when first seen. He looked in good health. On May 12 he had lost 2 lb., and did not appear so well. I cannot, however, detect any growth in the larynx.

Discussion.—Mr. W. M. MOLLISON asked how often it was intended to apply deep X-ray therapy. Dr. Watt, who carried out this treatment at Guy's Hospital, kept patients under observation for a long time, and gave a second dose three months after the first, whether there was a recurrence or not. The application of deep X-rays would rapidly get rid of such a growth as this. He (the speaker) had recently seen a case in which a large growth in the pyriform fossa disappeared in three weeks. The difficulty was to know just what cases would yield to deep X-rays, as the degree of malignancy could not be assessed. Two cases of growth in the post-cricoid region looked the same, but one responded to deep X-rays, while the other did not. He had one such case in which the patient was still well four and a half years after a similar application; there was no treatment either by diathermy or by surgery.

Mr. HAROLD BARWELL (President) said that a remarkable point about these malignant tumours was that one apparently limited growth would be most resistant both to rays and surgery, while another, quite formidable apparently, would be most amenable to treatment. It was for future workers to find a means of distinguishing these varieties before treatment. In the epitheliomatous type the microscope was a help; cases showing much tendency to keratinization were usually amenable to operative treatment, whereas those which did not show this tendency reacted well to rays, but tended to recur rapidly after operation.

Mr. E. BROUGHTON BARNES replied that to-day he was disappointed, as the patient showed some œdema, and there was none when he saw him last. It was hoped to arrange for further deep-ray treatment this month. His experience of the result of X-ray treatment alone was very limited. He thought that when there was a large mass of growth it was better to remove it by surgery, leaving a comparatively small mass to irradiate. In the present case the operation had been a palliative measure.

Tumour of Palate of Life-long Duration.

By O. POPPER, F.R.C.S.Ed.

X. Y., MALE, aged 54, gentleman farmer.

History.—Swelling on palate since birth. States that its size has not varied. Causes no difficulties with regard to speaking or swallowing. Advice sought on account of fear of possible malignant development.

Examination.—Hard swelling limited to hard palate in mid-line. Raised about ¼ in., and 1½ in. long. Tapers at its anterior end in the region of the anterior palatine foramina. Sessile on a broadish base anteriorly, somewhat undercut in its posterior quarter, which latter can be moved slightly.

106 Popper—Ibbotson—Dundas-Grant

Discussion.—Mr. HERBERT TILLEY said this was a very good illustration of the so-called "torus palatinus." A paper on the subject, by the late Sir Rickman Godlee, was read before the Section of Surgery of this Society.[1] At present he (the speaker) had a similar case, in a female, which he saw every year or so, in whom the torus was the size of half a walnut. It was as hard as stone and she says it has been there all her life but has caused very little inconvenience.

Mr. O. POPPER replied that he had wondered whether it was bony or cartilaginous. He thought the posterior and separate portion of the tumour was movable—an unusual feature in what was considered to be a pure osteoma.

Case for Diagnosis.

By WILLIAM IBBOTSON, F.R.C.S Ed.

PATIENT, a female, aged 39, married.

Symptoms: Dryness and pricking on left side of larynx for the past month, slight dysphagia, no dyspnœa, no night-sweats; patient losing weight.

The appearance in the larynx is unusual.

The patient is under the care of the local tuberculosis officer on account of pulmonary tuberculosis.

Sir JAMES DUNDAS-GRANT said he thought this was a case of tuberculosis; the co-existence of pulmonary tubercle would afford strong confirmation. He had seen a similar condition several times.

Hemiplegia of the Palate.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, a young man, was first seen a month ago, with "open" nasal speech and difficulty in swallowing, of three weeks' duration. The palate is drawn and dimpled to the right during phonation. There is no paralysis of the vocal cord, but a curtain movement of the pharynx (noted by Mr. Mollison). The only apparent cause was a severe attack of so-called "influenza" three months previously. His gums and throat were congested, but examination of a swab showed only cocci and no Klebs-Loeffler bacilli. The knee-jerks and ocular accommodation are normal. The exciting cause is probably sepsis and not diphtheria.

[1] *Proceedings*, 1908-09, ii (Sect. Surg.), 175.

Section of Ophthalmology.

President—Mr. Ernest Clarke, C.V.O., F.R.C.S.

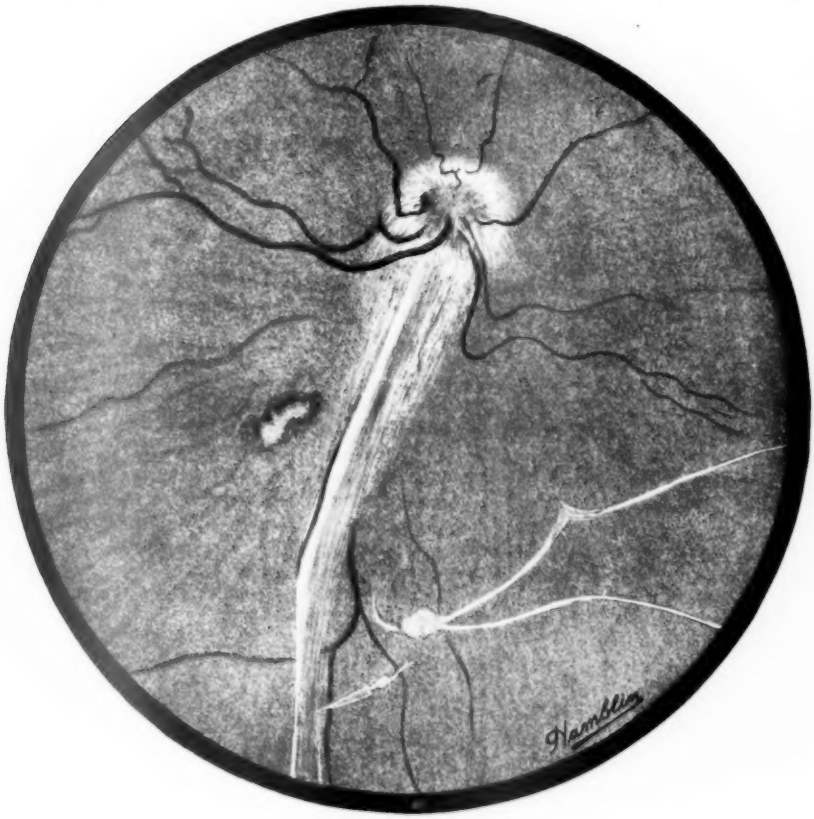
[*June 8th*, 1928].

CASES.

Case of Retinitis Proliferans without History.

By Norman B. B. Fleming, M.B.

This patient was brought up ten days ago. During the past year the right eye had been noticed to diverge frequently and the vision in that eye is only $\frac{6}{60}$. Vision in the left eye is normal. On examining the fundus I found the condition shown



Retinitis proliferans.

in the picture. There is a marked band of fibrous tissue, which stretches forward from the disc in the lower part of the fundus; it appears to be adherent to the fundus, not free in the vitreous. There are two other bands, with a mass of tissue adherent to them. In the fundus is one small patch of old inflammatory trouble. I

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could not elicit any history from the patient. It looks like retinitis proliferans, but there has been no serious injury during life, nor any injury at birth. The birth was normal, labour being conducted without the use of forceps. I shall be glad to hear the opinions of Members.

Mr. ERNEST CLARKE (President) said the condition looked like a result of an injury. Though there might not be a history of injury, children sometimes sustained injury and then forgot about it.

Arborescent Cataract.

By J. D. M. CARDELL, F.R.C.S.

THERE may be some connexion between the crystalline deposits in the lens and the fact that the patient, a man aged 70, is suffering from a blue-dot cataract. The deposit of the crystals has markedly reduced his vision, and the shape assumed by the formation is peculiar, as it originates in the posterior part of the lens, and projects forward into the substance of the lens like the branches of a tree. Crystalline deposits in the lens are fairly common, but the peculiar form in which they are aggregated in this instance makes the case interesting.

Right and Left Irido-Cyclitis.

By J. F. CUNNINGHAM, F.R.C.S. (for HUMPHREY NEAME, F.R.C.S.)

PATIENT, L. H., female, aged 41.

History.—May 21, 1928. Left eye bloodshot.

May 24, 1928. Right eye sore, painful and red. Vision misty for three days. No previous eye trouble. No recent illnesses.

Present Condition.—May 31, 1928. Pupils equal, active. Tension—right and left ciliary injection, marked in right, slight in left. Corneal haze, epithelial œdema, right > left. Œdema of substantia propria—right + +; left moderate.

H. and C.—Right eye, deep corneal striation. Dim view of fundus. No gross change. Left eye, fundus normal. No vitreous floaters. No iris vessels.

June 4, 1928.—Right: deep striation. Left: some fine keratitis punctata.

June 7, 1928.—Definite, very fine keratitis punctata on lower cornea right and left. Parallax apparently between the keratitis punctata and the folds.

Points of Special Interest for which the Case is exhibited.—In the right eye a complete circle of radiating folds of Descemet's membrane is present.

Neuro-fibromatosis or Plexiform Neuroma.

By FRANK JULER, F.R.C.S.

PATIENT, a male, aged 23.

History of increasing deformity since infancy.

The points of special interest consist in:—

(1) Fibromatosis of both upper lids and orbits, also of the right temporal and right occipital regions.

(2) Buphthalmos of the right eye.

Slit-lamp microscopy shows: (1) Plexus of nerves and vessels in right cornea. (2) Increase of nerves in part of left cornea.

Section of Surgery.

President—Mr. V. Warren Low, C.B., F.R.C.S.

[*March* 7, 1928.]

À Propos de la Colectomie pour Cancer.

Par Dr. T. de Martel (Paris).

Je veux d'abord vous dire les principes généraux qui me guident en chirurgie du gros intestin. Puis ces principes généraux exposés, quels procédés et quelles techniques en découlent naturellement à mon avis.

Un cancer du gros intestin existe souvent depuis longtemps quand il est reconnu par le médecin. À ce moment, la lumière de l'intestin est parfois très rétrécie au niveau de la tumeur et c'est fréquemment des accidents d'occlusion aiguë ou subaiguë qui attirent l'attention. Depuis longtemps déjà, l'intestin en amont de l'obstacle lutte contre cet obstacle. Il en résulte que sa lumière s'agrandit et que ses parois s'épaississent au-dessus de la tumeur. Au-dessous de la tumeur l'intestin est normal. Les deux bouts de l'intestin au-dessus et au-dessous de la tumeur seront donc très différents et se prêteront mal à une suture bout à bout. (Fig. 1.)

La tumeur est parfois fibreuse d'aspect, résistante au toucher, peu infectante, mais souvent aussi il s'agit de volumineuses tumeurs ulcérées, qui infectent profondément l'organisme qui les porte et qui provoquent dans leur voisinage immédiat de grandes modifications des tissus. Le mésocolon à leur niveau est œdématié, infiltré, bourré de microbes que la mobilisation opératoire de la tumeur mettra en mouvement et lancera dans la circulation en provoquant une septicémie. La paroi intestinale au-dessus de la tumeur participe à l'infiltration de la région. Elle est épaisse, œdémateuse, friable et se prête peu ou pas à des manœuvres opératoires. Les ganglions proches sont augmentés de volume et souvent purulents. Mais il ne faut jamais perdre de vue que tout gros ganglion qui accompagne un cancer n'est pas forcément un ganglion cancéreux, et il faut, si on veut faire utilement de la chirurgie du cancer, savoir se limiter dans l'ablation des ganglions.

Il faut, enfin, chaque fois qu'on réfléchit à la chirurgie du gros intestin ne pas oublier sa vascularisation qui est médiocre et se souvenir que les anastomoses entre les artères du gros intestin sont rares, et qu'en coupant une grosse branche colique on prive de sang tout un segment intestinal et qu'il ne faut jamais compter sur des branches anastomotiques pour suppléer l'artère manquante.

L'idée tout naturelle pour un chirurgien qui ignorerait ce que je viens de dire et qui se trouverait en présence d'un cancer de l'intestin, serait (1°) de décoller la tumeur de ses adhérences, de la libérer ; (2°) de couper l'intestin au-dessus et au-dessous de la tumeur et de l'enlever avec le coin de mésocôlon correspondant ; (3°) de rétablir la continuité de l'intestin par une suture bout-à-bout.

C'est ce qu'a fait Reybard avec succès en 1833.

Mais les chirurgiens qui ont voulu l'imiter ont souvent été moins heureux que lui.

En effet les deux grands dangers de la chirurgie du gros intestin sont la septicémie provoquée par la mobilisation de la tumeur, et l'infection due à la désunion des sutures, désunion très fréquente au niveau du gros intestin.

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Pour éviter autant qu'on le peut les accidents liés à la mobilisation de la tumeur, il ne faut y toucher qu'après l'avoir rendue aussi peu septique que possible et pour cela avant tout je pratique systématiquement un anus cæcal au moins huit ou dix jours avant autre opération.

L'anus cæcal évite la stagnation des matières au-dessus de la tumeur, il met l'intestin au repos en amont du cancer, il lui permet de reprendre son calibre et sa consistance habituels. Il rend possible les grands lavages de l'anus naturel à l'anus artificiel et vice versa. Sous son influence la septicité de la tumeur diminue, sa mobilisation devient moins dangereuse et plus facile, car les œdèmes purement inflammatoires rétrocèdent et les mésos reprennent de la souplesse. L'état général du malade s'améliore, la stercorémie disparaît, la fièvre, quand il y en a, diminue et souvent la température redevient normale. Le malade reprend du poids et mange. Ces avantages justifient pour moi l'anus cæcal que je pratique dans tous les cas.

D'ailleurs, très souvent le chirurgien est appelé pour la première fois auprès d'un cancéreux du côlon à l'occasion d'une occlusion aiguë, et, quand je me trouve dans ce cas, je pratique d'emblée l'anus cæcal. Je le pratique aussi quand je vois le malade avant tout accident d'occlusion pour les raisons que je vous ai exposées.

Voici la technique que je suis et qui m'a toujours donné de très bons résultats.

Je fais une incision de MacBurney dans la fosse iliaque droite. (Fig. 2.) Je cherche le cæcum, qui presque toujours très dilaté sort de lui-même. (Fig. 3A.)

Je le couche sur un lit de compresses que je glisse entre la paroi cæcale et les lèvres de l'incision, afin de protéger parfaitement la cavité abdominale.

A l'aide de pinces de Chaput qui fixent la paroi cæcale sans la blesser, je limite sur elle un petit quadrilatère là où elle bombe le plus. (Fig. 3B.)

J'incise le centre de ce quadrilatère, l'intestin se vide en jet. (Fig. 4A.)

Dès que l'écoulement se ralentit j'introduis dans le cæcum une de mes canules de verre olivaire prolongée par un tube de caoutchouc. (Fig. 5A.)

Sous les pinces de Chaput je noue un fil de lin qui fixe solidement la paroi cæcale autour de l'olive de verre. (Fig. 4B.) Je fais la toilette de la paroi cæcale à l'éther et à la teinture d'iode, puis je laisse rentrer le cæcum sans le fixer et je ferme la paroi abdominale. (Fig. 5B.)

Ce procédé que j'emploie depuis des années et qui me sert aussi pour la cholécystostomie, assure un drainage parfait du cæcum et permet ensuite de fermer la cæcostomie très facilement, ce qui est important. L'olive de verre et le tube de caoutchouc qui y est fixé ne se détachent pas avant douze ou quinze jours et permettent des lavages très faciles du gros intestin. La fermeture de cet anus cæcal se fait généralement spontanément dès que l'obstacle siégeant sur le gros intestin est supprimé.

Après une dizaine de jours de mise au repos et de lavages du côlon, je passe au second temps du traitement chirurgical de la tumeur. L'examen aux rayons X m'a généralement montré où elle se trouve et je n'ai pas à la chercher.

Il me faut alors choisir entre les différents procédés. Je ne crains pas de pratiquer un grand nombre d'opérations successives pour guérir mon malade et je sacrifie entièrement le temps à la sécurité.

Pour enlever une tumeur du gros intestin nous avons le choix entre différents procédés. Le plus simple est la colectomie segmentaire suivie de suture bout-à-bout du gros intestin. (Fig. 6.)

Ce procédé est commode pour le malade et pour le chirurgien puisqu'il a le grand avantage de tout terminer en une fois et je l'emploie volontiers, mais il faut que les conditions soient favorables, que le malade soit suffisamment résistant, que la tumeur n'exige pas pour son ablation le sacrifice d'une trop grande quantité d'intestins,

que les parois du côlon au-dessus de la tumeur ne soient pas altérées et friables et qu'on ait le sentiment qu'une fois l'ablation de la tumeur terminée les deux bouts coliques saigneront franchement : j'use toujours pour assurer la bonne irrigation des tranches du procédé de Lockhart-Mummery qui consiste à couper l'intestin très obliquement de façon à ce que son bord mésentérique soit plus long que son bord libre et qu'il reçoive plus de vaisseaux qu'il n'est nécessaire. (Fig 7.) Je fais chaque fois que je le peux une suture à trois plans, le premier plan muqueux, le second musculo-séreux, le troisième séro-séreux, mais cela n'est pas toujours facile et je me contente souvent d'une suture à deux plans, le premier comprenant toute l'épaisseur de l'intestin, le second séro-séreux. Je crois cependant cette suture très inférieure à la première. Cette colectomie segmentaire m'a donné de très bons résultats, mais j'en ai surtout usé dans les cas faciles.

Elle ne peut se faire quand il s'agit de tumeurs des extrémités du côlon.

Dans le cancer du cæcum je pratique, comme vous tous je crois, la seule opération possible, l'hémicolectomie droite suivie d'iléo-colostomie, que je ne fais pas termino-terminale, mais latéro-latérale. (Fig. 8.) Afin d'éviter la formation des culs-de-sac j'use du procédé de Souligoux qui consiste à fermer l'extrémité des deux intestins et à suturer ces deux extrémités fermées bout-à-bout. (Fig. 9). On fait alors tourner les deux intestins autour de cette suture et les appliquant l'un contre l'autre on pratique l'anastomose latérale. On obtient le résultat que voici, et plus tard une coupe longitudinale passant par l'anastomose se présente sous cet aspect.

Dans le cancer de la fin du sigmoïde la colectomie segmentaire est très difficile sinon impossible et je n'essaie jamais de rétablir la continuité de l'intestin. Je pratique l'ablation de la tumeur, la fermeture du bout inférieur et la mise à la peau du bout supérieur. (Fig. 10.) Là encore je sacrifie la beauté du résultat à la sécurité.

Je ne fais jamais la colectomie segmentaire en un temps si je vois que les parois intestinales sont en mauvais état, et je préfère user d'un procédé lent dont le procédé de Mikulicz est le type.

Le voici représenté schématiquement.

J'attire hors du ventre une longue boucle d'intestin dont la tumeur forme le sommet.

Je sectionne l'intestin aseptiquement à bonne distance au-dessus et au-dessous de la tumeur à l'aide de mon écraseur à trois branches. Je sectionne en coin le mésocôlon correspondant en vérifiant à l'aide de la diaphanoscopie que je n'ischémie pas les parties restantes du côlon. (Fig. 11.)

Je suture alors l'un à l'autre, les deux bouts fermés sur une longueur de six ou sept centimètres et sur une largeur de deux centimètres. Je les fixe dans la plaie, au péritoine pariétal. (Fig. 12.)

J'ouvre les écraseurs douze heures après.

Ce n'est que bien des jours plus tard que je ferme ce double anus en sectionnant l'éperon qui sépare les deux anus et en suturant l'une à l'autre leur demi-circonférence libre, par un surjet muco-muqueux recouvert par un musculo-séreux et par un séro-séreux. (Fig. 13.)

Ce procédé est long, mais sûr. Il est difficile à l'employer au niveau du côlon transverse chez les sujets gras à paroi épaisse. Je l'emploie très rarement.

Mais je tiens à dire que le procédé que je préfère qui présente à mes yeux le plus grand nombre d'indications est la colectomie subtotale suivie d'iléo-sigmoïdostomie et je l'emploie presque systématiquement. Sa technique est parfaitement réglée et l'ablation de la tumeur est faite aussi largement que possible. Voici comment je procède sans jamais craindre de perdre du temps et de multiplier les opérations.

Si le malade est maigre, encore résistant et en bon état, je pratique quelquefois

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l'opération en un seul temps, mais plus souvent je la pratique en deux ou même trois temps.

Dans un premier temps je sectionne l'intestin grêle à 25 centimètres du cæcum (Fig. 14), j'en ferme les deux bouts par invagination et je pratique une iléo-sigmoïdostomie latéro-latérale large. (Fig. 15.) Afin d'éviter les adhérences, j'enroule le bout cæcal du grêle dans une feuille de baudruche et j'en fais autant pour l'iléo-sigmoïdostomie, car rien n'est si gênant pour éxécuter le temps suivant que les adhérences que contractent les lignes de suture avec les anses intestinales avoisinantes. (Fig. 16.)

Dans un deuxième temps, je libère et ferme l'anus cæcal, je mobilise le côlon ascendant, je libère le grand épiploon de ses attaches à l'estomac et s'il s'agit d'un cancer du côlon gauche je sectionne le ligament colique gauche et je mobilise aussi le côlon descendant. Tout cela se fait très facilement et sans aucune hémorragie si on a soin pour décoller le côlon ascendant et descendant, d'érailler seulement la séreuse pariétale au bistouri et de terminer le décollement à la compresse montée.

Pour atteindre le ligament colique gauche il ne faut pas craindre de brancher une incision transversale sur l'incision longitudinale médiane. Je lie les vaisseaux coliques loin du bord mésentérique de l'intestin alors qu'ils ne sont encore que peu divisés afin de mettre le moins de ligatures possible : cependant quand j'approche du point où je sectionnerai l'intestin je veille à laisser complète l'arcade vasculaire correspondant à ce point.

Poir voir clairement les vaisseaux et leurs anastomoses et ménager une bonne irrigation de la partie du côlon que je conserverai, j'use de mon procédé de diaphanoscopie qui est souvent d'un grand secours.

Il consiste à éclairer les mésos par derrière à l'aide d'une lampe froide tandis que je fais l'obscurité complète dans la salle d'opération en éteignant toutes les autres lumières. Une seule pression du pied sur un bouton suffisant à obtenir ce résultat.

Quand le côlon est libéré jusqu'au point où je me propose de le couper je le sectionne aseptiquement à l'aide de l'écrasseur à trois branches (Fig. 17) et je mets hors du ventre le bout distal du côlon toujours fermé par l'écraseur. (Fig. 18.)

Je ferme la ligne d'incision et je suture soigneusement le péritoine pariétal au pourtour du côlon. Je suture les muscles et la peau et ce n'est qu'alors que j'ouvre l'écraseur.

Dans un dernier temps je ferme le côlon en restant extra-péritonéal.

Parfois cette mise à la peau du côlon en amont de l'iléo-sigmoïdostomie est difficile et les vaisseaux coliques sont tiraillés et je préfère dans ce cas fermer l'intestin en laissant la suture en dehors du péritoine, entre la péritoine et la paroi. Dans ce cas je pousse un gros tube de caoutchouc par l'anus naturel jusqu'à l'iléo-sigmoïdostomie pour tâcher d'éviter à ce niveau toute pression gazeuse.

Tels sont les principes qui me guident et que je peux résumer ainsi.

(1°) Ne jamais toucher à une tumeur du côlon sans avoir pratiqué bien des jours avant un anus cæcal.

(2°) Ne jamais craindre de diviser l'acte opératoire en plusieurs temps et sacrifier tout à la sécurité.

(3°) Se souvenir que pour que les sutures faites sur le côlon ne cédant pas il faut que la vascularisation de l'intestin ne soit pas compromise, et que ces sutures. pendant que se fait la cicatrisation soient soumises au minimum de pression.

(4°) Eviter d'abandonner dans le ventre une extrémité colique sectionnée et fermée par une suture. S'efforcer de la maintenir hors de la cavité péritonéale, entre le péritoine et la paroi musculaire.

Discussion.—Mr. J. P. LOCKHART-MUMMERY said he was almost in complete agreement with the views and principles expressed by Dr. de Martel. His own practice was to do a temporary cæcostomy at the time of operation and not beforehand except where a certain degree of obstruction was present. He thought this difference in practice was mainly due to diagnosis. Fifteen years ago a case of cancer of the colon before the onset of obstruction was very unusual, whereas now it was quite common to be able to diagnose the lesion before the onset of any obstruction. Perhaps things were not so advanced in France; the difference in technique might be due to this. He agreed with Dr. de Martel that where obstruction was present a preliminary cæcostomy was necessary, but where obstruction was absent he thought it was quite sufficient to perform a cæcostomy at the same time as the colectomy. The most important factor in successful colectomy was careful preservation of the blood supply to the new junction—he was very glad that Dr. de Martel agreed strongly with him in this.

With regard to technique, he preferred an end-to-end anastomosis by the open method. It was very important to protect the line of junction and he used a cuff of omentum, either free or attached, for this purpose. He was inclined to think it better than Dr. de Martel's gold beater's skin. The great difficulty of colectomy arose at the lower end of the sigmoid flexure. He agreed on the whole with Dr. de Martel that such cases are better finished by closing the upper end of the rectum and establishing a permanent colostomy. The bowel could be closed by the tube method, but although he had done this a good many times he thought the operation was attended by considerable risk from pelvic cellulitis, which was difficult to avoid. Some cases, however, were very successful and often patients were willing to run an extra risk to avoid the inconveniences of a permanent colostomy. He did not agree with Dr. de Martel as to the advisability of complete colectomy for growths in the left side of the colon. He believed these could be dealt with quite satisfactorily by local colectomy and that the after-results were better. Ileo-sigmoidostomy had not proved a very successful operation as regards the after-condition of patients.

[Mr. Lockhart-Mummery showed a number of slides demonstrating technique and diagnosis.]

Mr. GARNETT WRIGHT said that all surgeons agreed as to the necessity for preliminary drainage of the intestine in cases of carcinoma, when there was any obstruction. It was a curious paradox that, within limits, it was safer to remove a large portion of the colon than a small piece, because any anastomosis in the region of the ascending or descending colon was difficult and dangerous. For this reason he (the speaker) performed a right-hemicolectomy in all cases of cancer of the right side of the colon, anastomosing the terminal ileum to the transverse colon. In growths of the splenic flexure, descending colon and commencement of the sigmoid colon he removed the intestine from the middle of the transverse to the middle of the sigmoid colon. The anastomosis was thus made between two mobile parts of the intestine, which had a complete peritoneal coat. There was no tension whatsoever, and the suturing could be done entirely outside the abdomen. The dead space in the flank which remained after peritonizing the rear area was drained by a stab wound from the loin. If this was not done blood and serum would collect and become infected.

Instead of performing the cæcostomy at the times of operation he (Mr. Garnett Wright) preferred to pass a stomach tube through the anus a few inches beyond the anastomosis. There was a little difficulty in passing this tube through the rectum, but if it was guided by the hand of the surgeon in the pelvis the passage could be accomplished. Once through the rectum the tube could be readily passed any distance, and he had used it for anastomosis more than two feet from the anus. It was fixed in position by a stitch at the anal margin and removed in about three days' time. By means of this tube patency of the bowel was ensured from the beginning and flatus could readily pass. There was no distension, and therefore no strain on the suture line.

Mr. LIONEL E. C. NORBURY said it had been stated that persistent diarrhœa resulted in those cases of colectomy or hemi-colectomy in which the ileo-cæcal valve had been removed; he (the speaker) had removed the cæcum, or cæcum and ascending colon, in a number of

cases for atonic conditions. Diarrhœa had occurred in only one case, and this had been transient, lasting only for about ten days.

Again, in a large number of healthy people, with normally acting bowels, it had been proved by means of barium enemata that an incompetent ileo-cæcal valve was not uncommon.

DESCRIPTION OF ILLUSTRATIONS.

FIG. 1.—Au-dessus de la tumeur l'intestin est élargé et sa paroi épaissée. En aval de la tumeur l'intestin est normal. Les deux bouts de l'intestin, très differents, se prètent mal à une suture bout-à-bout.

FIG. 2.—Incision de MacBurney dans la fosse iliaque droite.

FIG. 3.—(A) Le cæcum très dilaté sort de lui-même. (B) Quatre pinces de Chaput limitent sur la paroi cæcale un petit quadrilatère.

FIG. 4.—(A) L'intestin incisé dans l'aire de la quadrilatère se vide en jet. (B) Sous les pinces de Chaput est passé un fil de lin qui fixera solidement la paroi cæcale autour de l'olive de verre.

FIG. 5.—(A) Le cæcum est fixé autour de l'olive de verre. (B) Le cæcum a été abandonné dans le ventre, et la paroi fermée.

FIG. 6.—La méthode de colectomie la plus simple est la colectomie segmentaire suivie de suture bout-à-bout du gros intestin.

FIG. 7.—(A) Section oblique du gros intestin suivant le procédé de Lockhart-Mummery assurant la bonne irrigation de la ligne de suture. (B) Suture du gros intestin terminée dans le procédé de Lockhart-Mummery.

FIG. 8.—Hemicolectomie droite suivie d'iléo-transversostomie latéro-latérale (procédé de choix pour le cancer du cæcum).

FIG. 9.—Procédé de Souligoux pour éviter la formation de culs-de-sac dans les anastomoses latéro-latérales. (A) Section de l'intestin et suture bout-à-bout des deux extrémités fermées de l'intestin. (B) Anastomose latéro-latérale des deux bouts de l'intestin. Le resultat vu sur une coupe longitudinale.

FIG. 10.—Cancer bas situé de l'anse sigmoïde. On n'essaie pas de rétablir la continuité de l'intestin. Fermeture du bout inférieure et mise à la peau du bout supérieur.

FIG. 11.—Section aseptique de l'intestin au-dessus et au-dessous de la tumeur à l'aide de l'écraseur à trois branches de T. de Martel.

FIG. 12.—(A i and A ii) Les deux bouts fermés de l'intestin sont suturés sur une longueur de 6 ou 7 centimètres et sur une largeur de 2 centimètres. Ils sont maintenus hors du ventre par les écraseurs et fixés au peritoine pariétal. Les écraseurs sont enlevés au bout de 24 heures. (B i and B ii) Section de l'éperon qui sépare la lumière des deux bouts de l'intestin premier temps du rétablissement de la continuité de l'intestin.

FIG. 13.—Bien des jours plus tard, l'éperon qui sépare la lumière des deux intestins a été sectionné (figs. 12 B i and ii) et la continuité de l'intestin est rétablie en suturant les deux demi-circonférences intestinales l'une à l'autre.

FIG. 14.—Section de l'intestin grêle à 25 centimètres du cæcum, et fermeture des deux bouts de l'intestin par rétournement (B).

FIG. 15.—Après section de l'intestin grêle, et fermeture des deux bouts, on pratique une iléo-sigmoïdostomie large.

FIG. 16.—Quelques jours après l'iléo-sigmoïdostomie on pratiquera la colectomie. Pour ne pas être gêné par les adhérences que l'intestin pourrait contracter avec les sutures elles ont été enveloppées de baudruche.

FIG. 17.—Le gros intestin est libéré jusques au-delà de la tumeur. Il va être sectionné à l'aide de l'écraseur à trois branches.

FIG. 18.—L'intestin sectionné au-dessus de l'iléo-colostomie est suturé au péritoine pariétal et maintenu fermé pendant 24 heures. Quelques jours plus tard, dans un dernier temps on fermera facilement cet anus.

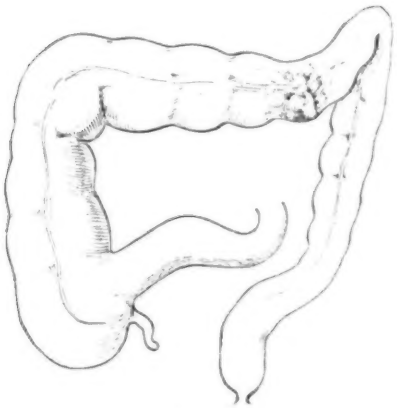

FIG. 1.

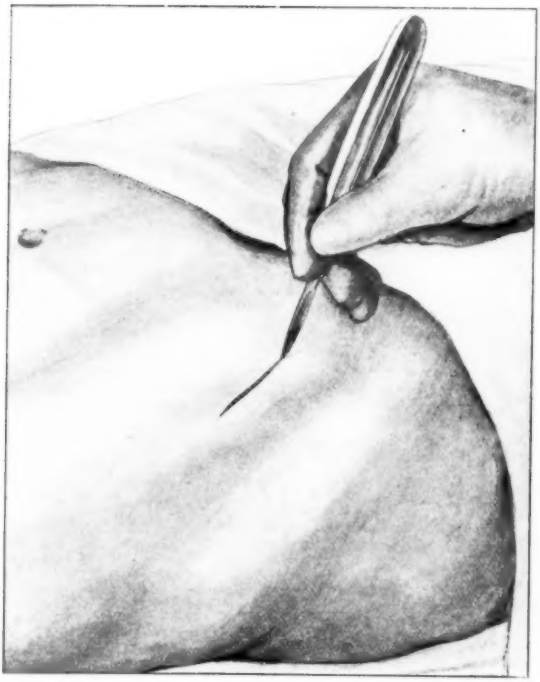

FIG. 2.

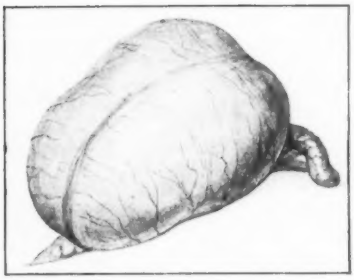

FIG. 3A.

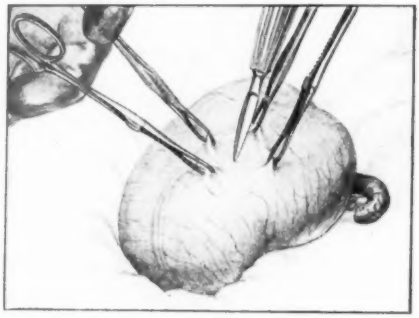

FIG. 3B.

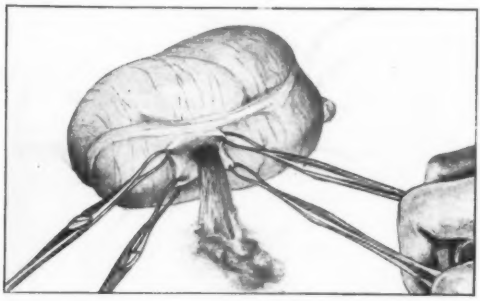

FIG. 4A.

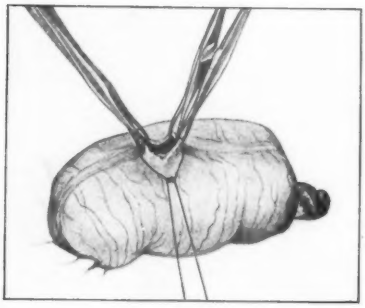

FIG. 4B.

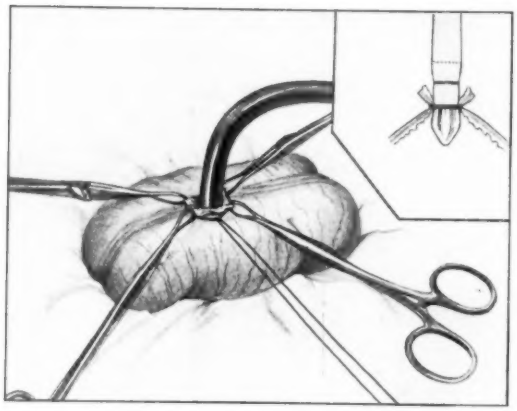

FIG. 5A.

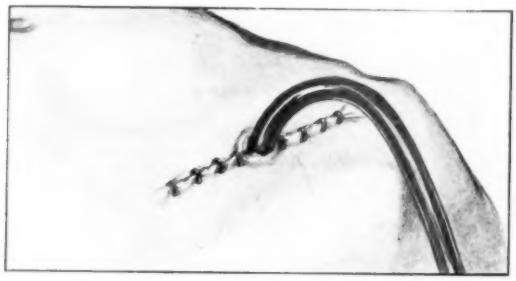

FIG. 5B.

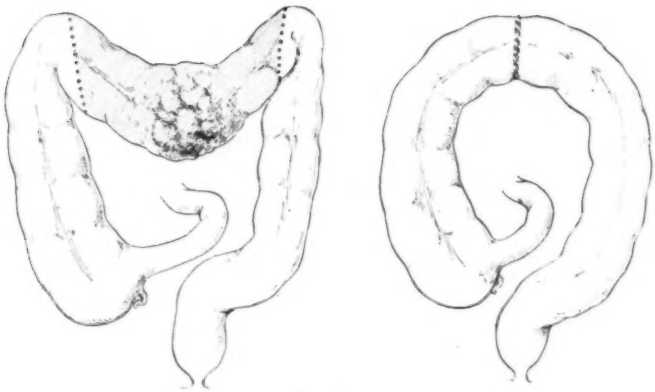

FIG. 6.

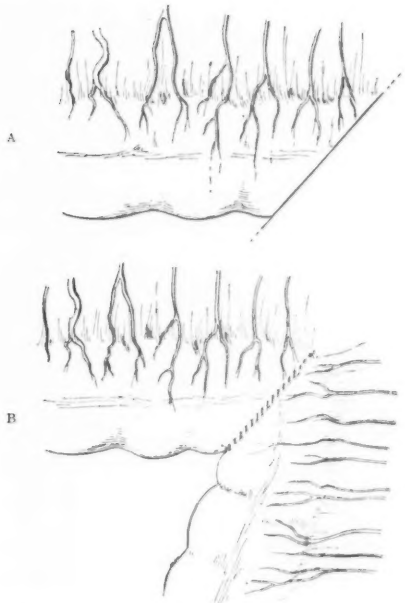

FIG 7.

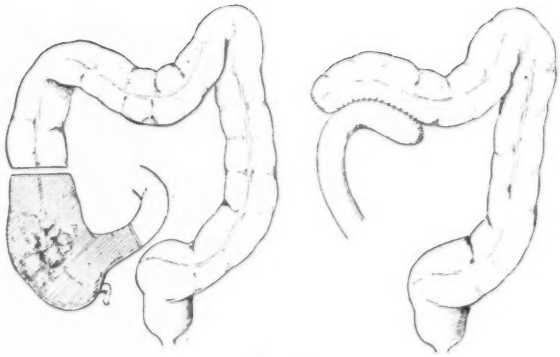

FIG. 8.

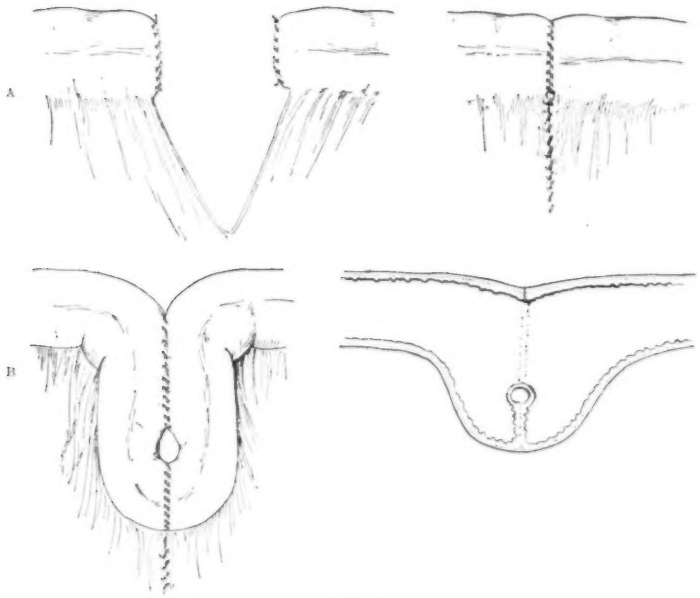

FIG. 9.

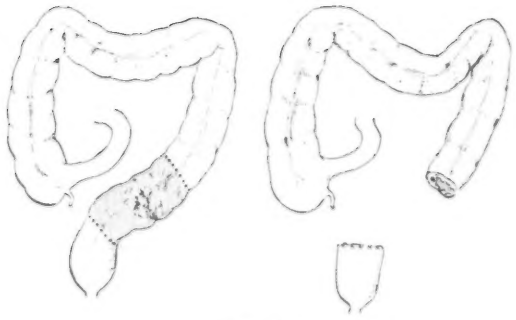

FIG. 10.

90 de Martel: *À Propos de la Colectomie pour Cancer*

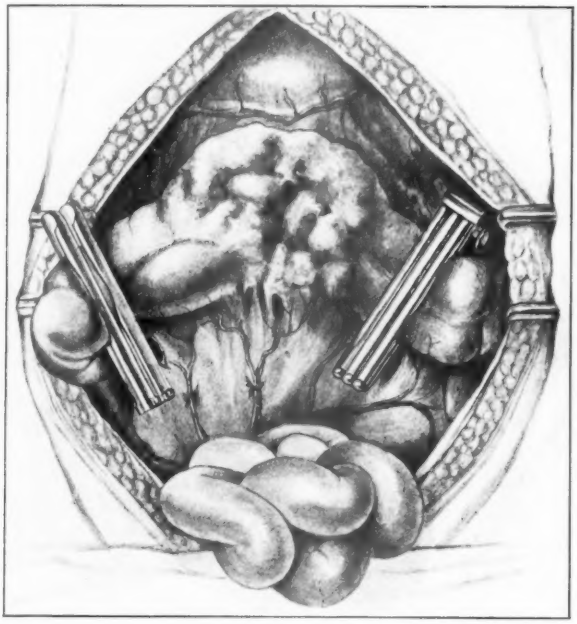

FIG. 11.

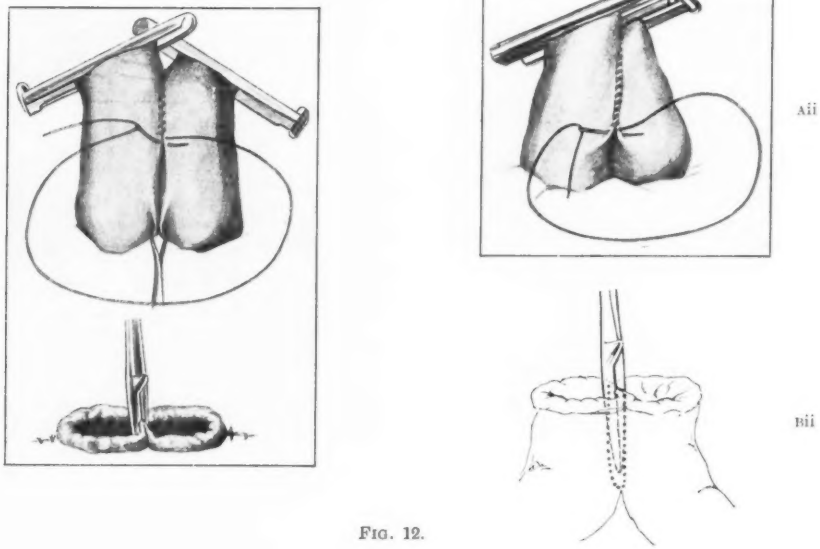

FIG. 12.

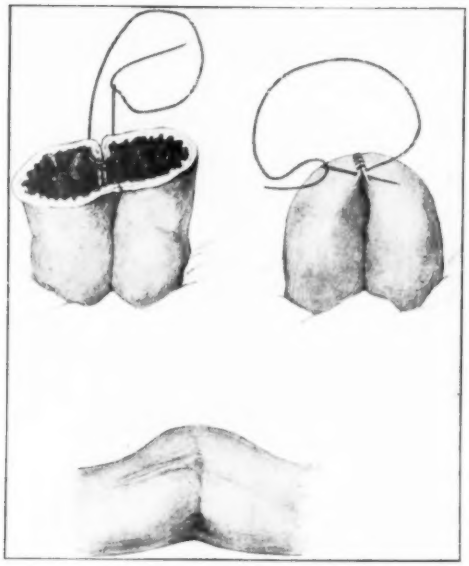

FIG. 13.

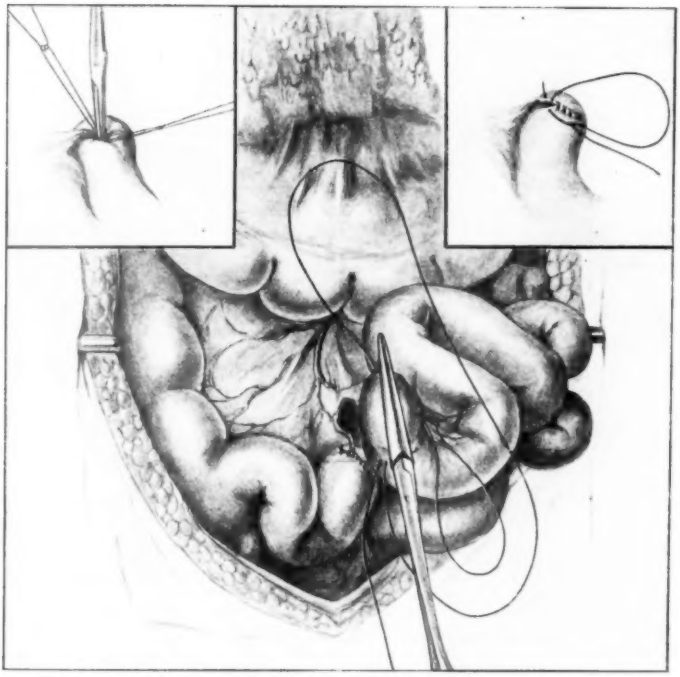

FIG. 14.

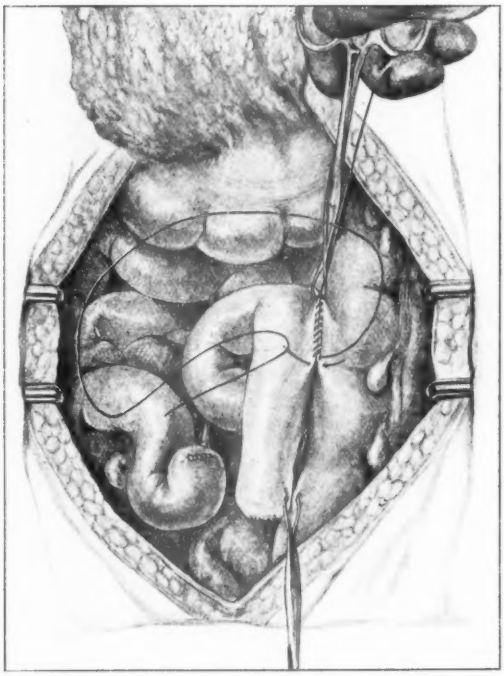

FIG. 15.

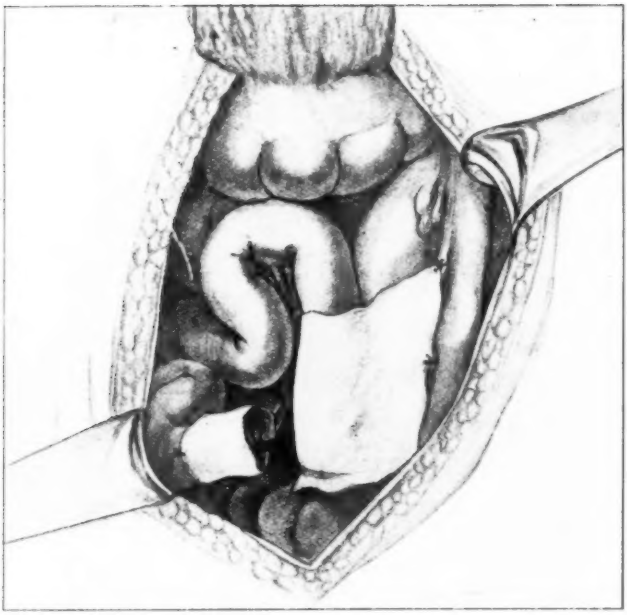

FIG. 16.

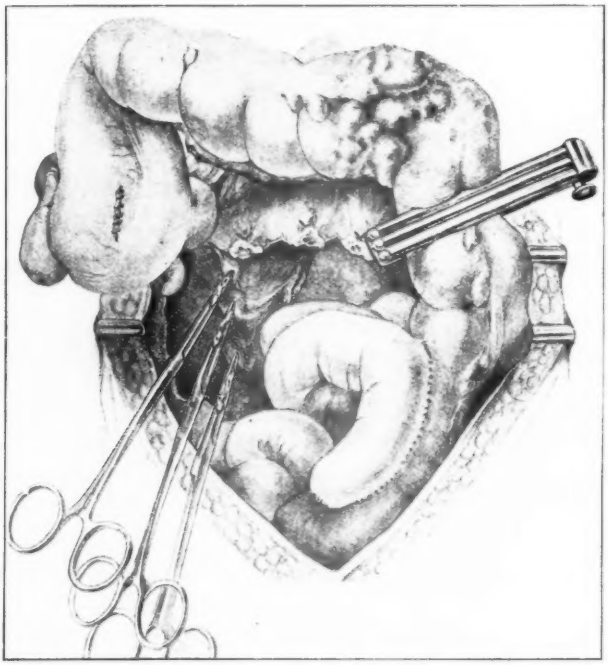

FIG. 17.

FIG. 18.

JOINT DISCUSSION No. 10.

Section of Dermatology and Section of Surgery.

[*May* 16, 1928.]

DISCUSSION ON THE TREATMENT OF VARICOSE ULCERS BY INTRAVENOUS INJECTIONS.

Dr. J. M. H. MacLeod (Chairman)

said that the treatment of varicose dermatitis and varicose ulcers by the injection of varicose veins, in order to occlude these veins by producing an artificial thrombo-phlebitis, was of great importance at the moment, not only to the Members of the Sections more immediately concerned with this discussion, but also to every medical practitioner. It was important for the surgeon, who, because of the uncertainty, and too often the disappointing results, of excision, must surely find the method a welcome addition to treatment; and it was important for the dermatologist who well knew the difficulties in the healing of varicose dermatitis and varicose ulcers, so long as the varicose veins remained patent.

This treatment was no new idea. All who had extensively practised the intravenous injection of veins knew that even normal veins might be occluded by the fluids used. This was particularly familiar to those who had treated malaria with intravenous injections of quinine, and, still more perhaps, to those who had had to treat cases of leprosy with intravenous injections of derivatives of various vegetable oils. So much had that been the case that one had frequently been obliged to adopt other forms of treatment because of veins having become occluded. A deliberate effort was made fifty years ago to cure varicose ulcers by the injection of perchloride of iron, and since that time all sorts of injections had been tried, such as iodo-tannin, chloral 1 in 40, and carbolic acid, but they had been given up because of the dangers associated with them, e.g., of embolism and severe necrosis, sometimes with fatal results. Recently, however, through the brilliant researches of Professor Sicard and his colleagues in Paris, this treatment, instead of being dangerous and uncertain, had been converted into a method of brilliant efficiency, by means of which excellent results were obtained, with the minimum of risk—in fact, with practically no danger; embolism was unknown in connexion with it, and anything like severe necrosis seldom occurred. It was a painless treatment, hence it required no anæsthetic, and had the tremendous advantage of not interfering with the avocation of the patient while it was being carried out.

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Professor SICARD, Dr. FORESTIER and Dr. GAUGIER (Paris).

CAUSES OF VARICOSE ULCERS.

Varicose ulcers develop on a ground predisposed by: (*a*) general vascular factors: (*b*) direct local factors.

(*a*) *General Causes.*—The presence of varicose veins, hereditary predisposition, age, derangement of the endocrine-sympathetic or vegetative system (the pituitary and ovarian principally, the thyroid and suprarenals playing a less important part). Syphilis, congenital or acquired, may exert an indirect action through the glandular disturbances, or it may have a direct local effect. General infections such as typhoid, or the various acute specific fevers, tuberculosis and cancer, do not cause varicose ulcers.

The action of these general influences on the veins of the lower limbs usually takes the form of a dual and local dystrophy.

(1) Weakness of the walls and valves of the vein; (2) weakness of the supporting perivascular connective tissue.

(*b*) *The Local Influences are Mechanical.*—These include intrapelvic pressure on the veins (old phlebitis, tumours, cysts, excessive abdominal adiposity, chronic intestinal stasis), constant standing, traumatism of the skin or bones, hæmorrhages and, most important of all, local infections of the skin, such as folliculitis and eczema, or of part of a vein, i.e., infective phlebitis.

THERAPEUTIC CLASSIFICATION.

The following classification is entirely based on the results of treatment. It is important to distinguish between those cases which have a previous history of phlebitis and those which have not.

(1) *Post-phlebitic ulceration* develops in an œdematous leg with more or less attendant venectasis. This form is not benefited either by surgery or obliterating injections, and can be treated only by rest, massage and careful support.

(2) *Dirty serpiginous ulcers* of long standing, surrounded by a peripheral sclerosis, phlebolithiasis and marked local dystrophy such as eczema, pigmentation, infection and folliculitis. There is sometimes an underlying periostitis which may be detected by X-rays. These conditions are also insusceptible to treatment by injection. They yield only to rest, disinfection, autogenous or stock vaccines, ultra-violet light and surgery (skin-grafting, sympathectomy or circular incision of the leg).

(3) *Ulcers with moderate or mild local disturbance*, which have no associated syphilis, endarteritis or arterio-sclerosis. These usually follow a patch of eczema, folliculitis, or pyogenic dermatitis. They may occur after the appearance of subcutaneous nodules resulting from phlebitis. Neglecting organo-therapy, the local treatment may be divided into two stages. In the first place the ulcer must be cleaned by means of ether, coal-tar, ichthyol, drying by hot air, ultra-violet rays, paraffin baths and elastic bandages. The second stage consists of the intravenous injections.

(4) *Mixed Syphilitic and Varicose Ulceration.*—This is sometimes difficult to distinguish from the preceding conditions. It is, however, usually marked by the relative smallness of the varicose area, the clearly defined ulceration, marked peripheral pigmentation, and the absence of infective dermatitis. It should be treated by the ordinary anti-syphilitic methods, together with the obliterative injections.

Treatment.

In dealing with ulceration, which is not consequent on phlebitis, the obliterative method which was introduced in 1916 by one of the present authors, gives excellent results.

We mostly use a solution of sodium salicylate in water, in strengths varying between 20 and 60 per cent. The injection should be made into the veins proximal to the ulcer, 2 to 3 c.c. being introduced in one dose. The actual location of the veins under the thickened tissues often requires very delicate and careful palpation, but during the course of the treatment the œdema subsides, and thus improves the demarcation of the varicose area. All dilated veins must be obliterated, and this is even more important in those patients showing ulceration than in those having simple varix. Obviously, the number and frequency of the injections, as well as the concentration of the solution, have all to be adapted to each individual case.

We have experimented with various other solutions, such as quinine, sodium citrate, hypertonic saline and glucose, every one of which has proved capable of causing obliteration of the veins and of bringing about gradual recovery. In fact, varicose veins are highly susceptible to this form of treatment. Their intima reacts easily and quickly to irritant liquids of any nature, whether caustic or anisotonic.

Even in those very early cases in which eczema is the predominant feature, the injections improve the skin condition as rapidly as they do the ulceration.

Conclusion.

The obliterative intravenous method is highly efficient whatever solution is used. It is particularly so when the local dystrophy is only moderate. We have treated many hundreds of cases with constant success and without a single untoward incident. Relapse is rare, but should it occur, further treatment along the same lines will always prove successful.

Mr. T. Twistington Higgins.

During the past two years, since visiting Professor Sicard's clinic, I (and those working with me) have been engaged in investigating the action of sclerosing injections, and have become increasingly impressed and interested in their value in the treatment of varicose veins in general.

Solutions Employed.

We have employed in our series of cases the following solutions:—

(1) Sodium salicylate in the usual 20 per cent., 30 per cent. and 40 per cent. solutions, as advocated by Professor Sicard. (2) Quinine and urethane. (3) Glucose, 50 per cent. We have followed the customary technique as to dosage and administration.

In our series we found that both sodium salicylate and quinine were very satisfactory. With glucose we have been disappointed. For the purposes of this discussion we have analysed 200 completed cases, and of these we find that thirty-five had active ulceration present at the time of treatment. This is a very small number, and we put forward our views with humility, suited to such a limited experience. From the cases we have treated we have formed the very definite opinion that sclerosing injections have been strikingly helpful in promoting the healing of many of these ulcers. On the other hand, in some the good effect has been less obvious. Our

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opinion is based on clinical impressions, i.e., subjective evidence, and is therefore very open to criticism. In all our cases the ulcers are naturally undergoing coincident treatment—with Unna's dressing, ointment and so forth—so that the intravenous injections are not the sole line of treatment in any case. Their precise value therefore must, it seems to me, always be a matter of opinion. Nevertheless, we have satisfied ourselves that in certain types of ulcer the effect of these injections is remarkable. Their limitations are not far to seek, if we keep in mind certain simple elementary considerations concerning varicose ulceration in general.

Factors in the Production of Varicose Ulcers.

The factors are: (1) Trophic, (2) traumatic, (3) infective.

(1) *Trophic.*—The long-continued circulatory disturbance produced by the varicosities leads to secondary changes in the skin, which I think we may correctly term trophic. It is possible that there is in some individuals a congenital susceptibility to degenerative changes in the veins and skin—possibly, as has been suggested by some authors, due to an endocrine defect. There seems some evidence of this in certain cases. At any rate, there is no doubt that these skin changes occur, and that they are progressive. Stages in the process can be recognized, beginning with alterations in the texture of the skin and going on to pigmentation and eczema. This may be described as the "pre-ulcerative" stage, and it is important to distinguish it as such. We recognize that patients in this stage are in danger of developing ulceration, and, indeed, ulceration may be regarded as the terminal stage in the degenerative process.

(2) *Traumatic.*—Some slight trauma is usually credited with the production of the ulcer. It may merely be the friction of clothing or the scratching of an irritating eczematous patch or the rupture of a venule.

(3) *Infection.*—Once the skin surface is broken, the final or infective element necessarily supervenes. Unhappily, in many of the cases seen, this is the predominant feature. Now it follows that the appearance of the individual ulcer will vary according to the extent and chronicity of the infection, so that at the one end of the scale we meet with a comparatively clean, purely trophic ulcer, and at the other the foul sore, bedded round with a dense area of cellulitis, in which the veins have become huge blood sacs which, when the limb is dependent, are thereby distended and the seat of severe burning pain.

Effects of Injections.

We should not expect injections of the veins to be equally efficacious in these varying types of ulcer and our experience bears out this expectation.

The type of ulcer which *does* heal rapidly with the aid of intravenous injections is that in which the trophic element still preponderates, i.e., in which a relatively small, clean ulcer occurs on a devitalized area at the distal extremity of a varicose column. The common situation for such an ulcer is over the inner aspect of the lower third of the tibia, in association with an internal saphenous varix. Artificial obliteration of the internal saphenous system in such cases produces a rapid healing of the ulcer; we have had several such cases, in which the ulceration has been present for long periods—even years—and all ordinary treatment, by Unna's method, ointment, etc., has failed to produce healing. The prompt response to the injections leaves one in no doubt as to their real value in such cases.

On the other hand, the infected ulcer presents a very different problem. It is necessary in the first place to use every effort to clean up the ulcerated surface by

means of fomentations, hydrochlorites or flavine dressings. Rest in bed is desirable in most of the cases, but is often very difficult to ensure.

Personally, in the case of a badly-infected ulcer, I prefer to delay injecting the veins until the area is reasonably clean in a surgical sense. I confess to a certain anxiety always in these cases; it seems to me that, if an embolism is ever to follow one's injection, it will be from a mild infection occurring in the clot, which permits some slight softening and a resulting separation, and I think the dirty ulcer must carry a certain potential risk in this way. Certainly in such cases I should always begin my injections well away from the ulcerated area at a higher level in the limb. The matter is further complicated by the fact that usually in such cases many injections are required and the veins are often very large—blood spaces with excessively thin walls; consequently perivenitis and necrosis are especially common, adding to the risk of infection. Nevertheless, despite all these anxieties and difficulties, in the case of the more desperate ulcers, we have satisfied ourselves that the injection treatment is of considerable value, though the effects are necessarily less immediate and obvious. We should certainly advocate their use, subject to the precautions we have indicated.

To sum up my views on the value of the fibrosing injections in the actual treatment of varicose ulceration: I am satisfied that this new method of treatment provides us with a valuable aid in counteracting the trophic factor, which really lies at the root of all varicose ulceration.

When the ulcer is purely trophic and uncomplicated by sepsis, the effect is prompt and most satisfactory. In the treatment of the septic ulcer the injections are also valuable, though certain precautions are desirable in employing them, and their effect on the healing of the ulcer is naturally more delayed.

In other words, the earlier in the life of the ulcer that we have the opportunity of applying this method of treatment, the better will be the result, and that brings me to my chief point.

I confess that I am much more interested in the *prevention* of ulceration than in its treatment. The real value of the injection treatment consists, to my mind, in its effects in the pre-ulcerative stages. I have already indicated that in certain cases, trophic changes in the skin—premonitory signs of ulceration—are easily recognized. I am confident that in some such cases we have averted the danger of ulceration by our injections. But to my mind the matter is far deeper than this, and I would like to indicate what, I think, is coming to be the position with regard to varicose veins in general, as a result of the introduction of this new treatment.

Hitherto surgeons have felt reasonably satisfied with the results of the operative treatment of varicose veins. The various operations which are in vogue, the Trendelenburg ligature excision and so forth, have, on the whole, given such satisfactory results that we have been liable to forget their limitations, though these are sufficiently obvious. Many people cannot or will not submit to an operation and all it involves. Others only do so when the varicosities have become extensive, and the necessary operative procedure is somewhat formidable and less likely to achieve a perfect result. So it comes about that of all cases of varicose veins occurring in the community, only a comparatively small percentage are submitted to surgical treatment at the most advantageous time. Some patients undergo operations at a later stage and are not infrequently disappointed by the result; while many certainly "carry on" in a philosophic spirit with the aid of elastic supports.

The result is that many patients only come before us for the first time when the more urgent complications, such as ulceration, have arisen. The "varicose ulcer" clinic has been for a long time a blot on our out-patient departments, and the amount of suffering and degradation produced by these ulcers is lamentable.

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But now comes this new method of treatment, and all our minds are necessarily stirred by the suggested possibilities of a completely altered outlook.

First, we must be satisfied that the method is really effective and that it is safe. If so, then since it is simple and involves no loss of work and no operations, we may reasonably hope that patients will be induced to undergo treatment in the early stages of the disease, and that the later complications, of which ulceration is the chief, will become increasingly rare.

First, then, as regards the question of safety. What are the risks from an artificially induced thrombo-phlebitis?

Embolism.

This is of course the bogey in all our minds. It seems to me that the weight of evidence is now such as to indicate that this is really a negligible risk. Professor Nobl has recorded 2,960 cases treated without any embolism. Sicard and his fellow-workers mention only one in ten years' experience, and in that case it seems clear that the embolism really came from a dilated right auricle and not from the site of injection at all.

In this country Alexander, Dunbar, Douthwaite, and recently Hanscell, Maingot and Hope Carlton, have recorded considerable numbers of cases with a similar immunity.

In our own series of 200 completed cases, involving some 1,232 injections, we have had no embolism.

The pathological evidence[1] shows that the characteristic reaction is primarily an irritation of the inner coat of the vein—"endovenitis" (Sicard)—and that such clot as may be formed is firmly adherent throughout its length. In a case of my own, in which the vein was excised on the seventh day after an injection with quinine, the clot was so firmly adherent that only by considerable force could it be detached from the wall of the vein.

Personally, I am convinced that the risk of embolism is exceedingly small, but I take two precautions:—

(1) I always endeavour to satisfy myself that there is no local or distal focus of sepsis from which the injected vein might become infected. I believe that the real risk of embolism lies in such a possibility.

(2) In cases under treatment, I advise the patient to avoid all excessive muscular activity, until such time as the process of organization is well advanced, that is, three weeks from the date of the last injection.

Perivenitis and Local Necrosis.

This is certainly a risk, which I think must always be present to some extent; the necrosis, when it occurs, is a local chemical gangrene and usually takes long in clearing up. In our series of 200 we noted twenty-three cases of perivenitis, and of these twelve went on to necrosis. The risk is greater in old patients and in bad cases. In mild cases and in young people the risk is extremely slight. Of our 200, we classified fifty-two as mild cases, and in these we only met with this complication in four instances. In two of these the necrosis was due to faulty injection and should not have occurred. This complication may arise in two ways:—

(1) From faulty technique—the irritant escaping into the surrounding tissues at the time of injection. This is clearly avoidable.

[1] Regard, G. L., *Rev. Méd. de la Suisse romande*, 1925, xlv, 102; Nobl, Löwenfeld, *Dermat. Wochenschr.*, 1925, xl, 1495.

(2) Even when the fluid has been accurately introduced into the vein, there may be such an excessive reaction that a perivenitis may develop, and this may progress to a necrosis.

Much may be done to avoid its occurrence, by care in dosage, especially in dealing with certain veins—notably those with very thin walls, and those directly overlying a bone—the front of the tibia or the malleoli and the internal femoral condyle.

General Toxic Symptoms.

These are unimportant. With sodium salicylate and glucose we have had none, though an occasional patient may become faint for a few moments from the needle prick. When quinine is used many patients taste the drug immediately after the injection, and a few complain of dizziness which soon passes off. The action of this drug on the uterus is of course important. One of our patients, a young unmarried woman, had severe abdominal pain and vicarious menstruation for two hours following an injection of 1½ c.c. quinine and urethane.

Results.

So much for the risks. What of the efficacy of these injections?

Provided the proper reaction is produced, the affected segment of vein is completely occluded and is ultimately obliterated as a venous channel. Underdosage or too protracted treatment may thicken the wall of the vein, without producing occlusion of the lumen, and such a vein cannot be said to be "cured"; we have had three such cases, which we classify as failures in our series. In each of these, for some reason, the course of treatment was interrupted, and this interruption probably explains the failure. It may be necessary to repeat the injections or to vary the irritant employed, but complete failure to produce occlusion of the vein is uncommon. The worst cases require many injections and much patience, but obliteration of the veins can be promised in practically every case. Secondary dilatations may occur at a later date elsewhere in the limb, as they so frequently do after operative removal, but these can be injected in their turn.

In conclusion, I think it may justifiably be claimed:—

(1) That obliterative thrombo-phlebitis, induced by intravenous injection of a suitable irritant, is simple, reasonably safe, and efficacious.

(2) That the procedure is a valuable aid in the treatment of varicose ulceration.

(3) That it is likely to prove more valuable still in the prevention of the condition.

Sir Sidney Alexander, M.D.

said he had practised this method since 1925, when he met Dr. J. Paraf, Professor Sicard's colleague, in Paris. Members might like to know whether there was any reason for using salicylate of soda or quinine, or whether both drugs acted equally well. He had used salicylate of soda solution in strengths of 20, 30, and 40 per cent., but he had never ventured to go to 60 per cent. He had also used quinine and urethane solution, and a solution of salicylate of soda, 20 to 40 per cent., with sodium chloride in distilled water, also salicylate of soda with urethane. His impression was that the best solution was salicylate of soda. It had the disadvantage that at the time it caused some pain and cramp, and in emotional patients it might lead to some faintness and giddiness. But it was easily tolerated, and these effects soon passed off. It also caused a quicker disappearance of the vein, the cosmetic result was better, and the thrombi formed were finer than those produced by the use of quinine. The disadvantage of

quinine was, that though there was no pain at the moment of injection, two or three days afterwards more severe pain was felt round the veins than that caused by the use of salicylate of soda. The immediate effect from the salicylate of soda was wonderful. Even before leaving the consulting room, patients would say the leg felt better. This was not often so after the use of quinine. As Mr. Twistington Higgins had pointed out, in the case of women quinine might have the disadvantage of causing metrorrhagia or bringing on the menses, or the patient might say she had strange pains in the abdomen, as if menstruation was about to commence. Quinine should not be used in cases in which pregnancy was suspected, otherwise a certain type of patient might come asking for veins to be treated and hoping for another result.

He, personally, had not had any experience regarding the injection of biniodide of mercury or of glucose. The massive doses of glucose suggested seemed to him objectionable.

The ideal posture during injection was the horizontal; but for practical purposes it was necessary to depart from the ideal. One must be certain that the needle and the solution actually got into the vein, and for this it was required to have the vein partly filled, which was not possible in the horizontal position, especially in the case of the smaller veins.

There were certain accidents which might happen in connexion with the method, and which one should try to avoid. Hæmatomata might form, owing to escape of blood from the vein. He (the speaker) used a transparent isinglass plaster placed over the puncture to give support to the vein and, by restricting the size of the clot, to improve the cosmetic result. Moreover, such a plaster was not easily seen through the silk stockings now so much worn.

In the treatment of varicose ulcers the recumbent position must be employed. One of the attractions of this new treatment was that it was ambulatory, but when the venous condition was complicated by varicose ulcers, one must insist on the patient lying up. In addition to injections into the varicose veins, local treatment must also be employed; he had found that cleansing with spirit and dabbing them with acriflavin, followed by the application of scarlet-red ointment, produced the quickest healing. Pressure by a cotton-wool bandage was also an aid.

Mr. David Levi.

My series consists of sixty cases of varicose veins and ulcers, treated by the method of injection advocated by Kantor, Professor Sicard and others. Three hundred treatments have been given.

At first glucose, and, later, salicylate was used, and at present quinine and urethane are the substances injected. The technique differs so little from that already described that I will pass on to the results.

In this series fifty-seven patients have been cured, by which I mean that all symptoms have been relieved up to the present time. Five have had thromboses at sites other than the site of injection, and three have not reacted at all and have been failures. In one of these cases, classed as a failure, a 66 per cent. solution of glucose, then a 25 per cent. solution of salicylate, and lastly, quinine and urethane, was used without success.

Many are familiar with the successes of this method of treating varicose veins by injection; I wish now to call attention to the failures. When a foreign substance is introduced into a varicose vein with the object of producing an obliterative endophlebitis, an endovascular clot is always produced during the early stages of occlusion of the vein. This clot is not confined to the site of inoculation; in 8 per cent. of my cases it occurred in the veins proximal to the site of injection, a

large mass appearing in the saphenous vein in the middle of the thigh within forty-eight hours of the treatment of the veins below the knee.

In some instances the veins appeared to allow the injected material to tranfuse through their walls into the subcutaneous tissues, causing a temporary swelling of the limb along the course of the vein injected, but in no instance was the sclerosing fluid injected into the subcutaneous tissues, and no sloughing or ulcer formation at the site of injection has been observed.

With the object of observing histologically the changes occurring in the vein, pieces of human saphenous vein have been excised at varying intervals of time following injection.

It is to the loose clot shown in fig. 3 that attention has been forcibly directed owing to catastrophes recorded in the literature by Oslon (in the *Journal of the*

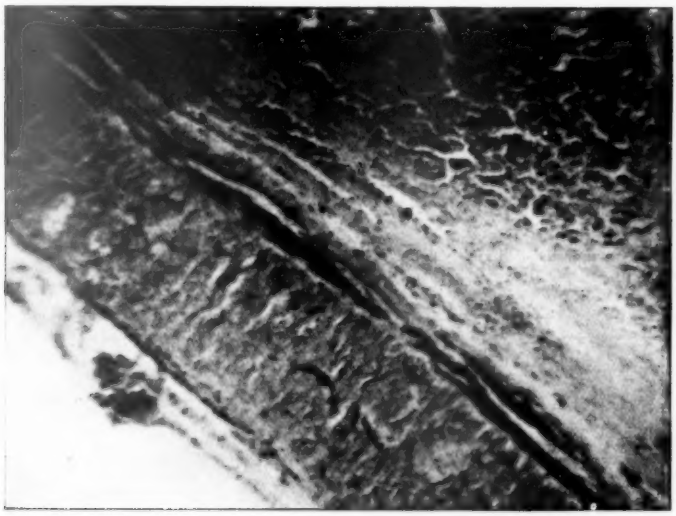

FIG. 1.—A vein a week after injection. There is a clot in the lumen in the early stages of organization. The endothelial lining of the vein has proliferated.

American Medical Association) and others. A pulmonary embolus developed in one of my cases. The patient had had three injections of a 66 per cent. solution of glucose. A pain developed in his side, with shortness of breath; he went to bed and in the morning spat up some blood; his doctor told him that he was suffering from pleurisy. I saw him at the end of a week when the symptoms had cleared; there was a clot in the saphenous vein, above the site of injection. The chest was X-rayed, and the patient was examined by my colleague, Dr. Copeman, who thought that there had been an embolus, although the signs at the date of the examination were negative. The patient recovered completely.

Another patient complained of a transient pain in the side. The incidence of embolus formation I am at present unable to judge; possibly clot may enter the venous circulation without producing severe illness, as it is caught by the lungs. If

FIG. 2.—A vein a fortnight after injection. The changes are more noticeable. Organization has proceeded and fibroblasts can be seen invading the central mass.

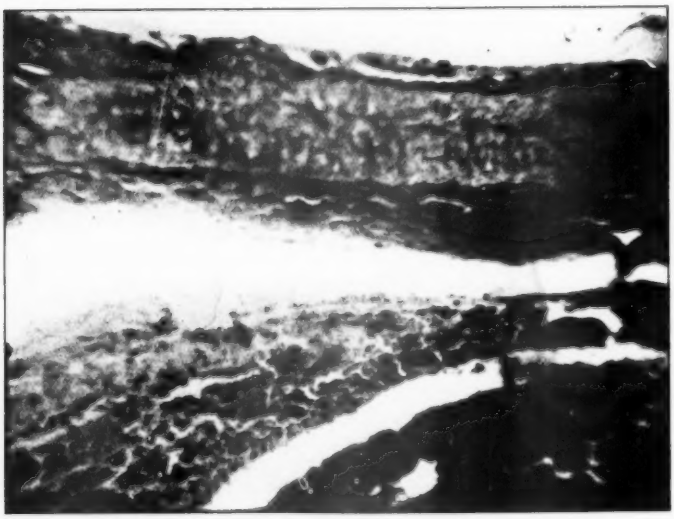

FIG. 3.—Another part of the vein shown in fig. 2, but under lower magnification, showing the clot separated from the vein wall.

it is not infected and lodges in an unimportant organ, a sudden transient pain may be all that calls attention to its presence.

As far as can be ascertained from a small series of cases, the use of glucose seems to be followed by more complications, judging by thromboses, than that of salicylate, while quinine with urethane appears to be the most innocuous injection that I have used.

With the object of studying more closely the factors concerned with the occlusion of veins, an attempt was made to produce an obliterative endophlebitis by injecting fluids into the ear veins of rabbits; the results were negative, as no clot or occlusion of the vein could be produced. The procedure was repeated in the presence of venous stasis, brought about by putting a tourniquet at the base of the rabbit's ear, but no greater success attended these efforts.

A diseased vein appears to be more susceptible to these injections than a normal vein; some patients react much more readily to them than others do, and it seems that the more diseased the vein the greater is the reaction.

I have to thank the Surgical Staff at St. Mary's Hospital for kindly allowing me to make use of their cases.

Dr. W. N. Goldsmith.

As all our methods of treatment should, as far as possible, be rational, we should endeavour to form a clear conception as to how far and in what way varices cause ulcers.

Trendelenburg showed that in the varicose state the *vis a tergo* was not enough to fill even the empty saphenous vein in the erect position. Magnus and Guleke, in 1921, demonstrated the centrifugal flow of the blood in varicose veins by means of Volkmann's hæmodromometer. In the horizontal position it is centripetal.

Through the centrifugal flow, blood, loaded with waste products, soaks back into the tissues from which the varix has arisen. This leads to chronic asphyxiation and a kind of atrophy. Œdema, or at any rate "latent œdema," is produced, as was shown experimentally by Frey: saline injected subcutaneously in the upright limb was retained indefinitely, and was only absorbed when the limb was raised to the horizontal position, and there was then an increased flow of urine.

The nutrition of the skin is further damaged by its being *stretched*, both by the veins themselves and by œdema. Dermatitis in the early stages is often seen confined to the skin just overlying the summit of varicose sacs. Perhaps this is the precursor of the peculiar ulcer described by Professor Sicard as associated with "venitis." In a recent article in the *British Journal of Actinotherapy*, Dr. W. J. O'Donovan argues that leg ulcers are not due to varices, because ulceration does not complicate varices in the abdomen or thigh. I think this is because in those situations the skin is not so easily put on the stretch by moderate œdema, and is not so much exposed to trauma. Moreover, many of the dilated abdominal veins are not true varices, but compensatory. It may also be objected that ulcers are not a common feature of cardiac or renal œdema. But in such cases the patients are usually in bed; further, the œdema is deeper; in varix the œdema is of the skin itself.

These underlying conditions prevent the skin from recovering normally from external injuries, whether mechanical, thermal, chemical or bacterial. But in "ulcus cruris" infection seems to play only a very minor rôle. Though Sabouraud calls it "chancre streptococcique," and Darier stresses the importance of staphylococci, Löwenfeld's thorough investigation of a large series of cases has shown the

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complete irregularity of the bacterial flora. Nobl concluded that the type and pathogenicity of the bacteria were of minor importance in comparison with the condition of the tissues.

Though of course ulcers of the leg occur in the absence of varices, they occur much more commonly in their presence; moreover, the varices always precede the ulcer.

It is therefore rational to expect the obliteration of the varicose veins to help the healing of such ulcers, and to diminish the likelihood of recurrence. It would not be rational to demand that obliteration of the veins should alone cure the ulcer.

A considered estimate of the effect of surgical extirpation of varices on the course of ulcers would be valuable, but unfortunately it is just in these cases that surgeons are naturally disinclined to operate, for fear of infection. The absence of this danger constitutes one of the great advantages of the injection treatment.

Of the many other advantages I shall only refer to one, namely, the possibility of ambulatory treatment. Prolonged rest in bed is to be avoided as far as possible, unless accompanied by massage and gymnastic movements, as the consequent weakening of all the muscles of the leg and its vessels renders them less than ever capable of dealing with the forces to which they will be exposed directly the patient gets up again.

My own experience is on a small scale, and any kind of statistics would be meaningless. Only in one case have I failed to obliterate the veins. There have been no undesirable complications, except one to be mentioned later. The effect on the general symptoms (complete relief of aching) has been excellent. Especially striking was the disappearance of œdema. In this connexion it is interesting to note that those local measures, which the majority of therapeutists have found most useful, are also designed to diminish œdema. Such are: compression by elastic bandage, by zinc-gelatin, or by rubber sponge and bandage, drying powders and Oppenheim's rhatany ointment, which acts by virtue of its tannic acid, and which I have found most successful.

The effect of the obliteration on the ulcers themselves is difficult to gauge. The ulcers in my cases have been relatively mild. Nearly all have healed up rapidly with simple local treatment without compression. Very likely some of these might have done so without treatment of the veins. But several had had about a year's local treatment, and only began to heal after the injections. In the cases that have resisted, there is advanced fibrotic contraction.

Of the large fibrotic ulcers I have no experience. They must be very difficult to heal by any methods, so that early treatment is especially imperative in order to prevent them.

How can one determine whether any particular vein should be obliterated or not? It is easy by means of two fingers, without Volkmann's hæmodromometer or X-ray screen, to determine whether the blood flows more actively upwards or downwards when the patient is standing. If it flows actively upwards, that vein should be left alone. I am surprised to find how many dilated veins *do* show an active upward flow. There is one possible fallacy here. When the lower finger is removed, it may appear as if the blood flows upwards when in reality the vein is merely filled from a higher column of blood through a collateral. But if the test is repeated at several levels and, each time, the vein fills up from the point whence the lower finger has been removed, one may safely conclude that there is an upward flow of blood in the vein.

The only other contra-indication is a present or past phlebitis.

I only make one injection at a time, as one never knows how far the effect will spread. On the whole, I prefer to begin distally, as I can then see clearly, after

a week, how far up the solidification has extended. If one begins proximally, there is a chance of a blood thrombus forming below the lower limit of the endophlebitis and not becoming securely anchored to the vein wall. At the moment of injection, and for a few minutes afterwards, the vein should be emptied of blood.

With regard to posture, the researches of Professor Sicard with lipiodol are very important. It is essential that the limb be at complete rest and quite flaccid, and the muscles must not be used in any way for half an hour after the injection.

With regard to Sir Sidney Alexander's point about "getting into" the veins, one can combine the two essentials by applying a tourniquet with the patient standing, thus filling the veins; the patient should then lie down and the foot should be supported above the level of the body. The needle is now inserted into the vein and the tourniquet removed. The blood is allowed to run out, and one can ensure the undiluted solution coming into contact with the vein wall by putting a finger on the vein above and below. The injection is now made, and immediately afterwards the leg is let down gently into the horizontal position.

My favourite preparation is 66 per cent. glucose, introduced by Nobl, Remenovsky and Löwenfeld (50 per cent., as used by Mr. Twistington Higgins, is not perhaps strong enough). It is non-toxic, scarcely irritating if injected into the surrounding tissues, and produces no pain at all. The only difficulty—a small one—is in the mode of sterilization. Theoretically, glucose must not be boiled, because, in boiling, it becomes partially decomposed. In actual practice I find that boiling for half an hour gives a solution that is most effective and absolutely non-toxic. But if it is carefully steamed for an hour on three successive days, according to bacteriological technique, it does seem to develop toxic properties, and I have seen it produce malaise and a very irritable general rash in three cases.

Occasionally glucose fails, and then I try 20 per cent. saline solution with some anæsthetic, as is now used by Linser, one of the earliest pioneers of the modern injection method. This solution is also completely non-toxic and is perhaps a little stronger in its action than glucose, but it causes more pain, being followed by a sensation of cramp. Further, if a small portion of this solution is accidentally introduced into the perivenous tissues, it produces very severe necrosis.

Sometimes I have used Génévrier's quinine solution. I have once seen it succeed when the other two had failed, but in other cases the converse might be true. It causes a great deal of pain; one of the patients had to stay in bed for several days. Minor tenderness lasts for weeks. Following its use, there have been reports of uterine cramps, accentuation of menstruation, onset of labour pains in pregnancy, scarlatiniform erythemata and severe symptoms in cases of idiosyncracy. I consider it too toxic for general use, especially in women.

I have not tried Professor Sicard's sodium salicylate. Nobl reported that its perivenous injection might produce uncontrollably severe reactions. In some persons stupor, headache and tinnitus occurred. Meisen found that in horses it produced clotting of the blood and only a rather short-lived endophlebitis. If endophlebitis clears up too quickly, there is some risk that obliteration will not be complete and that embolism may occur, but Professor Sicard's account of his experiences, both clinically and histologically, seem to indicate that my fears in this respect have been groundless.

To summarize my impressions: I am not convinced that obliteration of the veins is a method capable by itself of curing an ulcer, but I am certain that patients suffering from varicose ulcers are made incomparably more comfortable by that procedure, which also helps forward the healing of the ulcer by appropriate local measures and renders recurrence less likely.

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Dr. H. Beckett-Overy

said that since, two years ago, he had seen Dr. Gaugier apply this treatment, he had himself carried out over 500 injections, and his personal experience was distinctly in favour of sodium salicylate. He adopted Dr. Gaugier's method of inserting the needle with the patient standing, a posture to which very few patients objected, and he had not had any trouble in consequence. Immediately afterwards the patient was allowed to recline, and the injection was slowly carried out. It was possible to insert two or three needles at once and, after allowing the patient to lie down, to inject each successively, so long as an assistant placed swabs firmly over the punctures. In this way he could produce from 6 to 8 in. of venitis, and there was little reaction. He at once sealed the opening with "new skin," and then applied a bandage. He had carried out this method for six months, and in no case had he had trouble. In the case of some of the larger veins there was considerable swelling, but smaller veins disappeared completely, and in three to four hours the bandage was removed. The fact that salicylate of sodium produced pain was really an advantage, as any leakage outside the vein caused severe pain and warned the operator not to continue the injection.

Dr. St. G. Delisle Gray (Hove)

said he was induced to take up the injection treatment of varicose veins by hearing two opposed opinions on the subject, the holders of both views being men whose opinion he respected. One said it was a safe method, the other that it was dangerous on account of the risk of embolus involved. Boyd, in his "Surgical Pathology," a book which came out before the treatment of varicose veins was in vogue, drew a distinction between "clotting" and "intravascular thrombosis," stating that they were different processes. On microscopical examination, a clot of blood was seen to be composed of a network of fibrin, with red blood-corpuscles and white corpuscles entangled in it, the threads of fibrin radiating out from the platelets. When the endothelium of a vessel suffered surgical insult, as when a vessel was tied, or when the endothelium was damaged by injecting a sclerosing fluid, the platelets of the blood near the damaged endothelium were separated and deposited on the damaged surface, and there occurred a firm, pale thrombus with processes projecting into the vessel wall, rich in platelets (which, as it were, formed strengthening bolts), and in which there was no tryptic ferment present (as in infected thrombosis) to soften the clot, so that shifting was impossible, especially as varicose veins are serpentine as a rule. He (the speaker) generally found that varicose ulcers had a feeding vein coming in at the top, so that, by injecting that vein, one was preventing the stasis of blood in the ulcer. If a pipe in the earth was leaking, one could either dig down and mend the hole or turn it off at the top. The second procedure was that carried out in injecting veins for the treatment of varicose ulcers. He (the speaker) had found that sodium salicylate caused severe pain, though admittedly the results were excellent. He used quinine and urethane now, and always injected the patients in the standing-up posture. He used a super-saturated solution, and injected into the part two or three inches above the lesion; keeping the patient erect caused thrombosis to spread downwards, probably because of the greater specific gravity of the injected fluid. Professor Meisen (Copenhagen) said that varicose veins were largely due to toxins circulating in the blood, and that, in pregnant women, varicose veins often developed before there was any likelihood that the child was pressing on the pelvic veins. Professor Meisen also said that stasis caused the wall

of the vein to hypertrophy, and then to atrophy, and that this was the cause of the varicose condition. Recently a patient had come to him (the speaker) when she was a few months pregnant, and she had an enlarged vein. As an ulcer was threatening on the inner side of the ankle, he had injected into this vein sodium chloride, and immediately the whole vein had contracted. This experience supported Meisen's view that hypertrophy occurred first, enabling the vein wall, as in this case, to contract when irritated.

Dr. J. FORESTIER (in reply)

said that with regard to the causes of varicose veins, it was interesting to consider the endocrine dysfunctions; while the secretion of the suprarenal glands acted mostly on the arterial system, the secretion of the pituitary body acted chiefly on the venous system. As a local cause, attention must be paid to the early weakness of the supporting perivascular tissues, a fact which English doctors well recognized.

Two kinds of varicose ulcers must be recognized, one of which was amenable to injection treatment, the other not so readily amenable. In the first of these categories were the ulcers with moderate dystrophy, including those resulting both from varicose veins and syphilis. His colleagues and himself were not so positive as to which cases ought or ought not to be injected. The dirty serpiginous ulcers might cause alarm, but it was certainly sound to disinfect them, and then proceed to inject fibrosing solution. But a form of ulcer which should not receive the injection treatment was the post-phlebitic. Incidentally, he might point out that there were also varicose dilatations, not causing ulcers, which had not been included in the opening remarks. These venectasiæ of the abdominal wall and of the pubes were different from the ordinary varicosity under discussion, and should not be injected.

He fully agreed that the more frequently and the earlier varicose conditions were treated by injection, the less common would become the cases of dirty serpiginous ulcer, both in hospital and private practice.

In the matter of technique: he and his co-workers had not used the tourniquet; they did not see any advantage in doing so, and its use appeared to be due merely to custom or habit.

As to the posture of the patient: when he began working on this subject, seven years ago, patients were treated in the standing position. After the experiments by Dr. Gaugier with lipiodol, the lying-down posture was preferred, and he did not doubt that the results had since been better; the sclerosis had been more easily brought about, and the number of instances of secondary œdema due to fibrosis of the deep system was lessened. This treatment by injection was really one to be used by itself; he and his colleagues did not favour the supplementary ligation of the saphenous vein above.

With regard to the pathology: it was a pleasure to note that the speakers recognized the difference between chemical venitis and infectious phlebitis, especially as affecting the adhesion of the clot to the vessel wall. Mr. Levi appeared to be the only dissentient, and what he had said seemed to be supported by his pictures. One question which had not yet been solved was, whether the clot caused by sodium salicylate was of the same kind as that which followed the use of glucose.

As to possible accidents, he agreed that it was essential for the operator to be sure that his needle was well inside the vein, and he was glad Sir Sidney Alexander had insisted on this point. Since Professor Sicard and himself had discarded the use of sodium carbonate, they had not had one instance of slough caused by injection. In some cases there might be an apparent "slough," but this was not due to faulty

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technique. It arose from a special reaction of the vein rather far from the site of injection; a little spot of hæmorrhagic venitis where a nodule was formed under the skin. It did not come from the surface, as did a slough due to faulty technique.

Sodium salicylate caused only slight symptoms; those caused by quinine were more severe. He agreed that quinine should not be used if there was even a suspicion of pregnancy; there were cases in which abortion had followed the quinine injection.

With regard to the risk of embolism: Professor Sicard and his co-workers had now made over 300,000 injections of sodium salicylate and nearly 25,000 injections of quinine, and they had not one case of pulmonary embolism, occurring as an accident, to report. This immunity was largely due to the fact that before the method was employed in any case, the patient was subjected to a thorough examination as to the state of the urinary system, of the blood-pressure, and of the cardiac system, etc. The fatal cases reported in the literature had followed the use of glucose. He would not say that glucose was not good for the purpose, he would only say that he and his colleagues did not use it.

DEC 1 1928

VOL. XXI., NO. 12 OCTOBER, 1928.

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[*May* 2, 1928.]

Galen's View of the Vascular System in Relation to that of Harvey.

By J. PRENDERGAST, M.B.

GALEN was born in the year A.D. 130 at Pergamum, in Asia Minor, at a time when the last vestige of Greek liberty had expired, and when the Roman Empire had reached the zenith of its splendour. From his earliest youth his mind was formed and his opening genius moulded by the influence that Greece exerts on all her spiritual children. He spoke and wrote in the tongue of Hippocrates, Plato and Aristotle, and his works represent the last complete output of the scientific thought of antiquity, the culmination of all that had preceded.

He studied philosophy and literature in his native city, which had once been the seat of a great school and library. In pursuit of knowledge he also passed some time at Smyrna, Corinth and Alexandria, and finally migrated to Rome, which was then attracting, by its magnificence and the promise of reward, the most keen-witted and adventurous spirits from all the ends of its Empire.[1]

Here he rapidly rose to fame and came into contact with the Emperor and the highest officers of the State, many of whom frequented his demonstrations. To one[2] of them, who became so enamoured of dissection as to request a special treatise on the subject, we owe the present form of his great work on anatomy,[3] a tribute to the diversified interests that made up the intellectual life of that great city.

Galen had all the ardour of a restless and curious mind, and he followed all branches of knowledge with a zeal and competence that are truly astonishing. He may be regarded as the founder both of practical anatomy and of experimental physiology, for, though he had eminent predecessors[4] in both these fields, his works alone had that enduring quality which enabled them to survive the change of circumstance and the chance of fate. He is a voluminous writer[5] on medicine, on surgery, on materia medica and therapeutics, in which he employs a startlingly compendious array of ingredients, on history, philosophy, logic, metaphysics, ethics and several other subjects.

A brief survey of some of his anatomical and physiological works will help us to judge of the position which he is entitled to hold among those who have contributed, not merely to the advance of some particular branch of knowledge, but whose achievements have had an abiding influence on the general progress of mankind.

His description of the muscles is accurate and painstaking. For instance he describes the platysma myoides,[6] the interossei and lumbricals,[7] the muscles of the

1 Cf. Juvenal, Sat. III, lines 60 and 76.

2 Vol. II, p. 215. The reference by volumes is to Kühn's Edition in twenty volumes (in all twenty-two), 8vo, Leipzig, 1821-33.

3 Galen's great work on anatomy, *De Anatomicis Administrationibus*, is in fifteen books. The Greek originals of the last six and of part of the ninth have not so far been discovered. But Arabic translations in two manuscripts, once the property of Archbishop Marsh of Dublin, are preserved in the Bodleian. From these a translation into German has been made by Dr. Max Simon (Leipzig, 1906), and a French translation by C. V. Daremberg and M. G. Dugat. An English version has not yet appeared.

4 A list of the writers and authorities whom Galen quotes is contained in a tractate, *Material zur Würdigung Galens*, by J. Zimmermann.

5 He is said to have written some five hundred treatises. In his book *De Libris Propriis* he mentions one hundred and twenty-four dealing with purely philosophical subjects (cf. Clinton *Fasti Romani*, II, pp. 289-290). Of his surviving works which deal almost exclusively with medical subjects, ninety-eight are acknowledged to be genuine, nineteen are doubtful, forty-five are spurious and nineteen are fragments (Choulant, *Bücherkunde für die Aeltere Medizin*, 1841).

6 III, p. 917. 7 II, pp. 264 and 266.

80 Prendergast: *Galen and Harvey on the Vascular System*

larynx[1] and of the tongue, to which he ascribes sixteen.[2] He recognized the lacteals[3] which however had already been seen by Erasistratus. He described the ducts of the submaxillary and lingual glands[4] now connected with the names of Wharton and Rivinus. He recognized the cranial and spinal nerves, described the canal[5] leading from the third to the fourth cerebral ventricle, now called the aqueduct of Sylvius, the calamus scriptorius,[6] and the cerebral ventricles.[7]

His physiological investigations are no less revolutionary. He knew of what we call insensible perspiration, he ligatured the recurrent laryngeal nerves[8] and observed the results, he performed section of the spinal cord[9] at various levels and described the effects—the sensory and motor disturbances and the resulting incontinence—with an astonishing fidelity. He was familiar with the effect of cutting the nerve supply to a group of muscles on the action of the antagonists. He advised operating for cancer[10] and employed the catheter[11] for the very purposes for which it is employed to-day. He practised dissections and vivisections on all kinds of animals falling within the range of his investigations, with the exception of "gnats, flies, worms, bees and ants,"[12] which he considered too small for satisfactory examination.

Let us glance at some of the strange physiological beliefs that preceded him, doctrines which he quickly outgrew and rejected. Some believed that the voice came from the heart[13]; some, like his early teacher Pelops, that the blood-vessels originated in the brain.[14] Others, like Praxagoras,[15] thought the blood-vessels terminated as tendinous structures—beliefs which to us appear equally unwarrantable and untenable, though to men of that day parts of systems already venerable by long use and association. But the most interesting, historically, was the doctrine that the left side of the heart and the arteries, as that word itself implies, contained only air, or rather a special derivative of it, the "pneuma." That the arteries were empty after death seemed to lend this theory a certain *prima facie* probability. This doctrine was the basis of a good deal of the physiological and pathological teaching then current, and by Galen's day it had already enjoyed the sanction of at least four hundred years. He proceeded to show that this belief had no foundation in fact, and performed a number of experiments to prove that the arteries, like the veins, contained blood. On stabbing the heart he showed that blood immediately followed the withdrawal of the scalpel,[16] and that the scalpel itself was blood-stained, no matter how quickly the instrument was withdrawn. He opened an artery between two ligatures[17] and showed that this vessel contained only

1 III, p. 551. Galen frequently refers to dissections on the ape (πίθηκος) and advises his pupils to prosecute their anatomical investigations on the apes, which most closely resemble man (II, p. 223) with due regard to the difference in structure. Mistakes are made because dissectors forget that the structures found in a bullock, e.g., the heart, tongue and veins, are different from those found in man (II, p. 291). He states he is accustomed to work on apes suffocated in water, because this manner of death avoids the injuries to the neck caused by strangulation (XVIII, part 2, p. 928).

Haller, a very frank critic, in his *Bibliotheca Anatomica* (p. 83), says of Galen's dissections: "Multa etiam invenit rectiusque dixit, etsi passim, quoties plurima luce erat opus, utique prolixo sermone, quasi aliqua nebula se involvit, et veterum monumenta cum suis ita miscet, ut quae hominis sint monumenta, quae bestiae, non distinguas: inconstans etiam ut eandem partem corporis aliter et aliter describat." This is more or less true, but it is not very damaging to his reputation.

2 XVIII, (B) 959
3 II, p. 648. 4 III, p. 883. 5 III, p. 666. 6 II, p. 731. 7 V, p. 603 *seq.*
8 VIII, p. 268. 9 II, p. 676 *seq.*, cf. IV, p. 370 *seq.* 10 XV, p. 331. 11 XIV, p. 751 and 788.
12 V, p. 540. 13 V, p. 232.

14 V, pp. 527 and 544. Cf., *ibid.* p. 201, where Galen hints that the reason why the ancient anatomists thought that the blood-vessels originated in the brain was the presence of the large collection of vessels called the rete mirabile.

15 V, p. 188.

16 V, p. 184. A similar experiment was performed by Harvey, but with a different purpose, *Syd. Soc.*, Harvey, p. 27.

17 IV, p. 723.

blood, which immediately appeared as soon as the incision was made. He also insisted that, if the arteries contained only pneuma, an appreciable time would elapse before all the pneuma could escape through a wound in a vessel, particularly if the incision were small, and that the transit of this substance would delay the appearance of the blood. But his opponents had their answer ready, declaring that a disturbing factor underlay all these apparently effective demonstrations, for they claimed that on the first incision into the smallest superficial artery the whole arterial system was flooded with blood. But, he inquires, how is it that no obvious physical or mental change occurs in the animal, whose whole economy must be profoundly altered by the loss of the pneuma, on which, it was asserted, all physical and mental activities depended.[1]

Truly, they were a perverse generation "without shame in the presence of proof and conviction."[2] For Galen pressed home his conclusions with the ruthlessness of sincere and intense conviction. He had to contend against age-old prejudices, and provoked the hatred and animosity of his professional brethren, whose office it was to "keep trim the torch of ancient philosophy." This sense of personal injustice gives to much of his writings an eristic and contentious tone, which has been mistaken for mere combativeness and aggressiveness. Nevertheless, it must be admitted that Galen never reached that beatitude promised to those who rejoice when reviled and persecuted by men, far less to that which is promised to the lovers of peace.

Galen's demonstration that the arteries contain blood is a landmark in the advance of scientific thought. Yet the strange doctrine which it displaced, that only the veins normally contain blood, has particular interest, in that it suggested another question for which an answer had to be provided. The supposed transfer of blood from veins to arteries as the result of an incision demanded explanation. The explanation offered was that the terminal branches of the veins and arteries, too slender for detection by the naked eye, united together. This intercommunication was termed the *anastomosis*[3] of the vessels. It is one of the ironies, that are so often met with in the history of the sciences, that a doctrine, at once so fundamental and so remote apparently from positive proof, should have been deduced from an unstable and erroneous hypothesis. That it survived the overthrow of this hypothesis was no small matter, though in the course of time it gradually receded, and was at length almost forgotten.[4]

A vein is described by Galen as having one coat, an artery as having two, "namely an external one, which resembles that of a vein, and an internal one, which is thicker, and which possesses transverse (i.e., circular) fibres. The external coat has the same composition as that of a vein, that is, it has longitudinal fibres and some placed obliquely, but it is not furnished with transverse fibres. The thicker inner tunic of the artery has a lining resembling a layer of skin and very like a spider's web. It is mostly seen in large arteries and some term it the third coat of the artery."[5] This is a reference to the reticulation visible in the larger arteries.

1 IV, p. 715.

2 V, p. 184.

3 III, p. 92 *seq.* This communication of the vessels is referred to by the Hippocratic writer of the treatise Περὶ Ἄρθρων, Littré, Hippocrates, IV, p. 190, αἱ δε φλέβων καὶ ἀρτηρίων κοινωνίαι. But historically it is not easy to determine the value of this remark.

4 This doctrine of the anastomosis was still accepted in the School of Salerno, and the term "capillaries" is first employed by one of its writers, Copho, in his *Anatomia* "pars in sanguinem transit per capillares venas que vene gracillime sunt." In the magnificent illustrations of the *De Humani Corporis Fabrica*, Vesalius makes no attempt to portray the anastomosis. Caesalpino believed in the anastomosis of the vessels but in a manner in which neither Galen nor modern physiologists understand the term. It was not until Malpighi with his microscope demonstrated the capillaries of the lungs (*De Pulmonibus*, 1661) that this question could be declared definitely settled.

5 II, p. 601. Cp. III, p. 468.

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The difference in appearance between the blood in the arteries and that in the veins was emphatically recorded. The contents of the artery were described as escaping rhythmically [1] when the vessel was severed, while the blood of the veins simply poured out.

Minute and detailed instructions are given for exposing the heart [2] without injuring the parietal pleura, so that the mechanism of respiration may be preserved intact during observation. "And when the heart is exposed," he continues, "we suggest that you observe all its movements without disturbing it. You will notice that the animal continues to breathe, and can make a noise, and if you set him free you will see that he can run about as well as ever. And if the wound is stitched up, he will take food and drink when he wants it." [3]

Galen records his observations very thoroughly:—

"You will see that both ventricles pulsate in the same way, and not the left only, as some people imagine. . . . As time goes on, the ventricular contractions grow more feeble. Then the right ventricle is seen to act with a rhythm of its own. And this will be particularly noticed when all motion begins to fail. At first the parts at the apex cease to beat, and so on until movements persist only at the base of the heart. And when this also ceases to beat, some languid and feeble movements separated by increasing intervals of rest persist in the auricles." [4]

Unfortunately, he came to the conclusion that the *diastole* of the heart and arteries took place simultaneously. Perhaps it is some justification to plead Harvey's early difficulties with this problem, when he "found the task so truly arduous" [5] and almost concluded with Fracastorius "that the motion of the heart was only to be comprehended by God."

In his description of the valves [6] of the heart much is borrowed by Galen from the Alexandrian school, and he freely acknowledges his obligations to Erasistratus. Herophilus, it appears, was less happy in his description of these structures. The number,[7] shape, position and attachments of the valves are given accurately, and he adds the explanatory remark (for Galen never sins through lack of exposition):—

"In using the term 'tricuspid,' reference is made, not to the shape of each leaf, but to the appearance they present when pressed together during closure, for during this time their whole outline resembles that of a three-edged spear, and this name, therefore, is suitable for the valve at the opening of the vena cava. No one has given an appropriate name to the segments at the opening of the arteria venosa (pulmonary vein)." [8]

The coronary vein [9] is described as encircling the heart like a crown, and having its origin outside the auriculo-ventricular ring. The origin and distribution of the coronary arteries [10] are also carefully set down. He points out that the apex of the

1 VIII, p. 5. V, p. 106. *Ibid.*, p. 537.
2 II, p. 626, *seq.* 3 II, p. 631. 4 II, p. 640.
5 Text by T. Willis, issued by the Sydenham Society, p. 19. All subsequent references to Harvey are given to this edition.
6 The first reference to the valves of the heart is found in the Hippocratic treatise Περι καρδίης. See Littré Hippocrates, IX, p. 86. By some this is accounted a spurious work. Aristotle does not mention the valves.
7 III, p. 476, *seq.*
8 III, p. 478. It may be remarked that Galen, like Aristotle, regarded the right auricle as part of the vena cava. The left auricle he regarded as belonging to the pulmonary system of vessels (arteria venosa) and the left auricle ventricular orifice was designated the opening of the arteria venosa. Vesalius was the first to use the term "mitral" to describe this valve. *De Humani Corporis Fabrica*, 1555, p. 740.
9 III, p. 477. Cp. II, p. 618.
10 II, p. 618. The branches of these arteries are followed with extreme care, and they are shown to preserve their character as blood-vessels throughout, a demonstration which goes to refute the doctrine of Praxagoras who held that arteries normally became converted into nerves, V, p. 189.

heart is conterminous with the wall of the left ventricle, and notices that the interventricular groove may sometimes be so well defined as to suggest a kind of division of the organ.

The character and functions of ordinary muscle[1] Galen apprehended clearly. He describes a muscle as consisting of a head, body and tail, the latter ending normally in a tendon attached to some bony structure.[2] He dissected and traced the attachments of the subcutaneous muscles, such as the *constrictor oris*, *orbicularis palpebrarum* and the sphincters, and these obviously differed from the long muscles in the manner of attachment and in mode of action, but they all had this in common that they were under control of the will. The reaction of the heart was different. Section of its nerve supply,[3] that is, of the branch of the vagus, was not followed by cessation of its activities, its beat continued uninterrupted, and its pulsation continued even when the heart was removed from the body.[4] This freedom from voluntary control and this independent activity showed a characteristic not recognized in any other muscle.

"The heart is composed of firm flesh and it is almost insensitive to pain. It is built up of many kinds of fibres (*ἶνες*), and, though it thus seems to bear a very close relationship to ordinary muscle, nevertheless there is a marked difference between the two structures. For the fibres of ordinary muscle are disposed in one set way, that is, they are either longitudinal or transverse. . . . But the heart has longitudinal, transverse, and, in addition, oblique fibres."[5]

"The flesh of the heart is not of a simple character, for it contains fibres like those of muscle, and to those the flesh is bound. Besides, the nature of the fibres is not quite similar, for in muscles they are associated with tendons and ligaments. But the fibres of the heart are of a special kind, like those of the coats of the arteries and veins, or those of the stomach, intestines, womb, and of the hollow organs."[6]

There is conclusive evidence that Galen appreciated the special nature of the cardiac musculature, though he hesitated to call it muscle, a term which he reserved for the ordinary voluntary muscles. This distinction he expressed as clearly as it was possible in his day. Not until the advent of the microscope could clearer definition be given. His views do not diverge widely from those of modern physiologists. The difference is mainly one of terms.

We now come to a physiological doctrine which, above all others, is associated with Galen's name, the doctrine of the communication of the two ventricles of the heart by foramina in the septum. This fateful dogma[7] became embodied in physiological teaching, and during the centuries that followed it acted as a stumbling-block to inquiry. Heroic efforts were required to shatter it. Vesalius led the way, and at length Harvey came on the scene, and the ancient doctrine disappears for ever. But by this time another important contribution to scientific knowledge had been made, of which we find no traces in Galen, that the veins had valves[8], and this made the final solution much easier.

1 This statement should be qualified by the remark that he believed that the nerve which supplied the muscle became assimilated with the tendon and with the fibrous structures of the muscle, which he apparently regarded as the more important element in muscle. Cf. Aristotle, *De Part. Animal.*, III, 4, and *Hist. Animal.*, III, 5.

2 IV, p. 374, *seq.*

3 II, p. 614.

4 *Ibid.* Aristotle also made this experiment on the heart of a tortoise, *De Respiratione*, XVII, 479a.

5 III, p. 437.

6 I, p. 601. Cp. II, p. 610.

7 This transfer of blood is hinted at, obscurely and diffidently, by one of the Hippocratic writers. Littré, Hippocrates. *Περὶ Καρδίης*, IX, ch. xi, p. 88.

8 On the history of this discovery a very exhaustive and luminous study has been made by Dr. K. J. Franklin in a paper which he read before this Society last year.

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The postulation of the interventricular foramina was the outcome of a purely mechanical view of the heart's activity. Of the two vascular orifices on the right side of the heart, that of the auriculo-ventricular opening which led blood into the heart, was larger than that of the vena arteriosa (pulmonary artery) through which the blood passed out. Therefore, it was regarded as inevitable that during contraction of the heart some blood should be forced through the small pits or depressions of the septum into the left ventricle. The supposed inverse disparity of the vessels on the left side of the heart, the larger opening of the aorta requiring more blood than could be supplied by the smaller auriculo-ventricular orifice, seemed to lend corroboration to this conclusion.

An appeal to teleological principles,[1] a method of reasoning employed by Galen, always dextrously but often perversely, strengthened the grounds for this argument. In a perfect scheme of things ordered by an all-wise Providence, the pits were not fortuitous arrangements, but created according to design, and to serve a purpose. It is that design and purpose that Galen feels called upon to unfold!

Galen's theory of the penetrating foramina may have received confirmation from the actual finding of patent foramina ovalia in the hearts of lower animals.[2] I have come across no reference in his works to such an experience, but with his large range of investigations it is not improbable that he met with such cases. That he failed to record them need excite no surprise, for his interest was concentrated on what is habitually and normally found in man. "Man," with Galen, "is the measure of all things."

To the question, Did Galen in any measure grasp the principle of the general circulation? we can confidently answer "No." It is true there are statements in his writings which seem to show that he referred to the flow of arterial blood in one direction. But he did not correlate these observations, or blend such knowledge into a harmonious whole. In one passage he states that "the arteries receive more from the heart than they return, and the reason for this is the action of the valves."[3] He refers to the wounds sustained by hunters in the chase, or by soldiers in battle, and remarks on the numbness and coldness that set in in the parts beyond, very quickly if the artery were wounded, slowly if the vein alone were injured.[4] Another passage that adumbrates a faint realization of the centrifugal flow of blood is the one in which he refers to the effect of a tight bandage on the limb, when the distant part becomes cold and livid "because it is deprived of the heat which flows down from above through all the parts."[5] And another

1 Teleological arguments were the legacy of antiquity, and may be regarded as an indispensable apparatus of the early researchers. Thales, Anaximenes, Plato, Aristotle, all explained problems whose real solution baffled them, by an appeal to teleological explanation, resting on the principle, unavowed or acknowledged, "that nature made nothing in vain." Besides, there exists in all minds a desire for æsthetic completeness which impels them to fill the gaps in their systems by the best devices they can muster. W. D. Ross, commenting on this tendency in Aristotle, is of the opinion that "in these and many other cases the teleological method has enabled him to detect facts which no mechanical treatment would have discovered." But that Aristotle was not slavishly bound by this principle is shown in the well-known passage on bees (*De Generatione*, III, p. 10, 760b). And many passages could be quoted from Galen which show him to be discriminating and judicious in relating function to structure. For instance, when treating of the use of the prostate and seminal vesicles, he says, "such is the opinion we hold about their use, and it will serve us until we discover by some scientific means their real purpose. Perhaps the uses we have suggested are quite correct, and yet it is possible that a more accurate account of their function may be discovered" (IV, p. 649).

2 Cf. Harvey's reference to persisting foramina ovalia in the goose, snipe and various birds and many of the smaller animals . . . and in one of the larger members of the mouse family," *Syd. Soc.*, Harvey, p. 38; cf. Dr. Bell Pettigrew in the *Transactions of the Royal Society of Edinburgh*, 1864, p. 781, where he refers to the openings in the hearts of serpents. Galen records that he dissected serpents (II, p. 537). Dr. John Davy in his *Physiological Researches* (1863, p. 334) describes the foramina in the heart of an alligator in words which read like a transcript from Galen's pages.

3 V, p. 166. 4 V, p. 160. 5 *Ibid.*

hint of the Harveian doctrine is in his description of the operation of inserting a reed into the artery:

"It is evident that if we do the experiment in this way no blood can flow from the artery, since the upper part, the direction from which the blood flows (*τοῦ μὲν ὑψηλοτέρου μέρους, ὅθεν ἔπιῤῥεῖ τὸ αἷμα βρόχῳ διειλημμένου*) is held by a ligature."[1]

And he quotes the famous passage from Plato about the heart, "which is the fountain of the blood, which is rapidly carried round through all the limbs."[2] But he is here dealing with the same question that Plato is discussing, the seat of the "irascible soul," and there is really no emphasis on the blood or its movements.

On the other hand, many passages might be brought forward which would make it abundantly clear that Galen did not apprehend the principle of the circulation of the blood. One or two short references will be enough. The theory that "it was the purpose of the arteries to draw something from the veins by means of the anastomosis which existed between them,"[3] and that "the arteries which anastomose with the veins attract from them the lightest and finest part of the blood which they contain"[4] precluded the consideration of anything like a continuous movement in one direction.

It is not such details alone, but his general view of the blood-system, that made a conception of a circulation impossible to him.

The vascular system was compared to an irrigation system,[5] such a common feature of Eastern agricultural method. Fields and gardens are kept productive by damming streams and diverting water into channels cut to suit the lie of the ground. When one section of a field is watered, by which time it presents the appearance of a sodden expanse, the water is diverted to another. And so this goes on in sequence and in series. "And a similar arrangement is found in the bodies of animals. Numerous canals distributed throughout the various parts of the body bring them blood in the same way as the water is carried along in the garden. And the spaces between those canals have been wonderfully arranged by Nature, so that they are well provided by blood, which they attract to themselves, and they are never in danger of being overcharged with blood running in at an unseasonable time."[6]

Arterial and venous blood had separate and distinct rôles in the animal economy, and each was associated with a different kind of pneuma. The arterial blood supplied organs of finer texture, as the lungs, the venous blood served the needs of the heavier organs, as the liver.[7] The tissues had the power of attracting the blood most suitable to their requirements by a kind of specific attraction, and, under the influence of this vital stress, the blood might move centralwards, or towards the periphery, as need demanded.

A word about Galen's views on respiration. He declares that, if we could discover why flames are extinguished when covered by an air-tight vessel, we would have a clue to the nature of the substance gained by the bodily heat through inspiration.[8] It was obvious, too, that something of a special kind or quality was

1 II, p. 646.

2 V, p. 573. In another place, *ibid.*, p. 581, he changes the word *μέλη* to *μέρη*. Cf. Plato, *Timaeus*, XXXI. Someone, for whom the comprehensiveness of Plato has not been spoilt by familiarity, proves from this passage that Plato understood the circulation of the blood!

3 III, p. 512. 4 II, p. 204.

5 This simile is used by Plato (*Timaeus*, XXXV), and by Aristotle (*De Part. Animal.*, III, 5) to illustrate the same idea.

6 II, p. 211. 7 III, p. 495. 8 IV, p. 487.

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absorbed from the inspired air, and this could be demonstrated by the simple experiment of holding the breath in deep inspiration, when, although there was plenty of air in the lung, the subject of the experiment suffered acutely.[1]

Part of the air, he believed, underwent a change in the lung.[2] About the amount of the aerial substance that entered the heart, Galen is obviously unsettled and undecided, for in one passage he admits "that very little indeed, or none at all of the substance of the air, ever reaches the heart."[3] But a kind of mixture of both blood and air (*ἐξ ἀμφοῖν τι μικτὸν*) was sent out from the left side of the heart[4] by the aorta. To prove that the air taken in by inspiration was not transferred directly into the heart he artificially dilated the thorax,[5] which was then seen to remain expanded and incompressible, an experiment repeated by Harvey[6] to demonstrate the same truth.

The arteries too had a respiratory function, the superficial arteries drawing in air during the active phase which he regarded as that of diastole, and expelling the useless products of vital activity during systole. The air absorbed was for the cooling and feeding of the vital heat.

This doctrine, to which Galen gave acquiescence, had its foundation in the very beginnings of Greek science, and it may be referred back to the speculations of Anaximenes,[7] and to popular primitive physiology. It is found in a high state of development in Empedocles,[8] and amongst the Hippocratic[9] writers, where it takes a more or less similar place in pathological causation as that found in the Alexandrian school. It had its foundation in the mystery that surrounded the nature of air. The idea of the possibility that the atmospheric air could be absorbed through the skin had no doubt its basis and confirmation in the well-known phenomenon of the absorption of drugs by inunction.[10] This belief survived till Harvey's day.

In demolishing this ancient theory Harvey is completely captain of his soul, and in a few telling phrases he shows the unsubstantial foundations on which it had so long rested.[11]

The idea of the pulmonary circulation is clearly outlined by Galen. And to this teaching Harvey found it convenient to turn when dealing with the reactionaries of his day, those who would "admit nothing unless upon authority."[12] Harvey uses the advocacy of the "father of physicians" very skilfully, showing himself a master in dialectic and an artist in appeal. Galen points out that a transfer of blood from

1 VI, p. 479. 2 III, p. 540. 3 IV, p. 725.

4 III, p. 547. 5 III, p. 548. 6 Harvey p. 16.

7 Burton's *Early Greek Philosophy*, p. 75, *seq*.

8 Aristotle, *De Respiratione*, VII, 473b. Aristotle himself does not subscribe to this doctrine.

9 Cf. Littré, Hippocrates, *Περὶ Ἱερῆς Νούσου*, IV, p. 368. VII, p. 372, et alibi. Hippocrates uses *ἀήρ*, *πνεῦμα* and *ἄνεμος* for the breath without apparent distinction.

10 Cf. Harvey, p. 72. "It will not, therefore, be found unreasonable, perchance, if we say that the veins, by means of their orifices, absorb some of the things that are applied externally, and carry them inwards with the blood."

11 Harvey, p. 10, *seq*. 12 Harvey, p. 42.

the branches of the pulmonary artery to those of the pulmonary vein takes place, and that this blood reaches the heart. But this doctrine is deduced from an indefensible hypothesis about the action of the pulmonary valve. This valve was supposed to remain closed during the contraction of the thorax, during which time some of the blood is pressed out from the arteries into the veins through the anastomosis of these vessels in the lung, and the force of the propulsive power of the heart is completely underrated.

It is a piece of brilliant speculation, but Galen fails to grasp its real or relative value. It would be unwise to stress too much the importance of it in his physiological system. It is enough to admit that he outlined the path by which the pulmonary circulation could be effected.

This short sketch would not be complete without a word about his observations on the changes that take place in the heart immediately after birth. One of the most interesting passages in Galen, and one that puts him in the forefront of original investigators, is that in which he lucidly describes these alterations and adaptations. Others, like Aristotle, had observed its beginnings and growth, but it was left for Galen to study its development and subsequent evolution more completely. In speaking of the fœtal heart he draws attention to the opening, called the foramen ovale [1] by later anatomists, through which the right communicated with the left side of the heart. He refers to the canal which leads from the vena arteriosa (pulmonary artery) to the aorta, which we call the ductus arteriosus. He describes what is now termed the Eustachian valve, and its purpose and function are clearly defined. He continues:—

"All these operations of Nature are indeed wonderful, but more wonderful still is the subsequent occlusion of this opening (foramen ovale). For soon after birth, within a day or two, or in some cases within four or five, or even occasionally after a longer period, the valve which guards this opening is found to be uniting with the orifice, but not yet completely adherent.

"If you inspect the same place in a fully grown animal you will find it so well-formed, that you would hesitate to think that there was a time when it was pierced through. . . .

"In the same way, the duct that connects the aorta and the vena arteriosa (pulmonary artery) although all the other parts continue to increase and grow after birth, this, so far from keeping pace with the general development may be seen to become thinner and more slender, and in the end to dry up and disappear altogether." [2]

It is not perhaps too much to assert, that for accuracy and descriptive narration of what happens at this period of the animal's history, Galen has not been excelled.

1 This opening (le trou Botal) is associated with the name of an anatomist who has been claimed as its discoverer. He flourished, however, some fourteen hundred years after Galen's death.

2 IV, p. 243, *seq*.

88 *Section of the History of Medicine*

During the Session 1927-1928, the following papers have also been read at meetings of the Section :—

[*March* 7, 1928.]

"Louis Pasteur and Fermentation." By Mr. F. PRESCOTT.

[*May* 2, 1928.]

"Harvey's Work on Generation." By PROFESSOR F. J. COLE, D.SC.

Section for the Study of Disease in Children.

President—Mr. GEORGE E. WAUGH, F.R.C.S.

PROVINCIAL MEETING HELD AT LEICESTER, JUNE 30, 1928.

The Intensive Method of Serum Treatment of Diphtheria.

By H. STANLEY BANKS, M.D.

(*Superintendent of the Leicester Isolation Hospital and Sanatorium.*)

THE important clinical features of the type of case treated by this method by the author since April, 1927, are as follows:—

(1) Extensive diphtheritic membrane involving tonsils, fauces, uvula, soft or hard palate, and naso-pharynx; ill-defined, glassy membrane towards the spreading margin was of grave import, as was also œdema of fauces.

(2) Cervical adenitis and peri-glandular œdema of severe degree, the swelling varying in size from that of a hen's egg to that of the extreme "bull-neck" type.

(3) Nasal discharge, especially if hæmorrhagic; membrane in nares.

(4) Fœtid odour from the throat, typical of severe diphtheria.

(5) Severe toxæmia characterized by delirium and restlessness or by great lethargy and hebetude; and evidenced in some cases by petechiæ in the skin and by submucous hæmorrhages.

In several cases of the hæmorrhagic type in the series the patients had recovered.

The average dosage of anti-toxin employed for the above type of case was 70,000 units intravenously and 30,000 intramuscularly. As much as 250,000 units had been given to a case. As far as possible, the whole of the anti-toxin needed was given at one time, but if, after a lapse of from twelve to twenty-four hours the patient's response was not sufficiently good, a similar large dose was administered. The criteria of adequate dosage were (1) disappearance of toxic symptoms within twelve to twenty-four hours, brightness of demeanour replacing delirium or hebetude, and (2) opaque appearance of the membrane and well-defined edges within the same period.

In 300 verified cases of diphtheria treated during a period of twelve months, there were twenty-seven cases of the severe toxic type described above, with six deaths. The hospital death-rate for diphtheria was reduced from an average of 9·3 per cent. over the previous decennium, to 2·6 per cent. during the twelve months period under review.

Since commencing the intravenous intensive method I have learned that V. Bie, of Copenhagen, has employed similar methods since 1920, and, dealing with about 1,500 cases a year has secured a mortality-rate of 1·3 per cent. Laryngeal cases, and cases dying within twenty-four hours of admission, being excluded from the figures.

[Lantern slides were shown illustrating the severe toxic type of case and the method of treatment.]

94 Lawson: *Congenital Malformation of Pelvis & Lower Extremities*

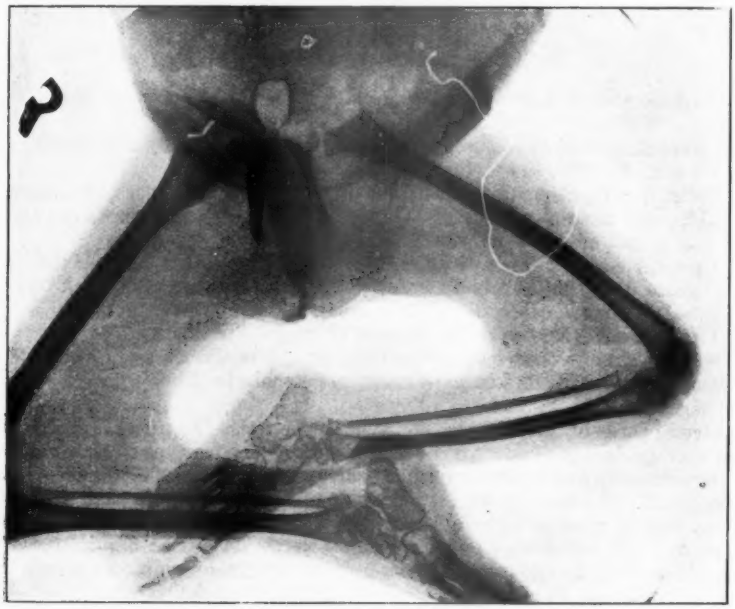
CASES.

The following cases were shown:—

By ROBERT S. LAWSON, F.R.C.S.

(I) Congenital Malformation of Pelvis and Lower Extremities.

E. W., FEMALE, aged 3 years. The child was born with this marked deformity of the hips, both legs being widely abducted, and lying in the frog position. There was a curious bony prominence in the centre of the dorsal region of the spine, but no definite spina bifida occulta could be seen. The child has an extra thumb on the right hand. Both knee-joints are deformed, and there is a marked dimple over each trochanter.



Congenital malformation of pelvis and lower extremities.

(II) Raynaud's Disease.

C. W., A BOY, aged $2\frac{1}{2}$ years. This child's feet have always been cold and blue, and about the beginning of March, 1928, what seemed to be chilblains developed over the third toes. These became swollen and painful; the skin peeled off, and the toes were found to be black and ulcerating underneath. There is a history that, occasionally, the toes became quite blanched before gangrene set in. Skiagram shows no definite spina bifida. Wassermann reaction, negative.

As it was thought advisable to try the effect of peri-arterial sympathectomy, this was carried out on the femoral artery on the left side, on March 28. A month later, the toe on the right side was completely healed, while the slough on the left side had not yet completely separated, showing that the peri-arterial sympathectomy apparently had no effect on the condition.

(III) Osteogenesis Imperfecta.

G. C., A GIRL, aged 12 years.

About six weeks ago she went to her doctor complaining of pain in her back On examination, the spine was found to be rigid in the lumbar region. She was sent to the infirmary, where I saw her as a case of early Pott's disease. The radiographer reported that the bones showed an irregularly mottled appearance, but no definite caries. The blueness of the sclerotics suggested the diagnosis of osteogenesis imperfecta, and, on making inquiry, it was discovered that she had fractured several of her bones at various periods of her life, and also that her father had had seven fractures before he was 6 years old, including fractures of both arms and both legs. Of five brothers he was the only one who suffered from brittle bones. His mother—that is the child's paternal grandmother—is supposed to have died from the same disease, but no definite proof could be obtained of this. In the light of these findings, it is probable that this patient has sustained a fracture of one of the vertebræ, which caused the temporary rigidity of the spinal muscles. This fracture cannot be shown by X-rays, but the films indicate the defective ossification of the bodies of the vertebræ—a defect common to all the other bones. Old fractures can be made out in the region of the olecranon and the right tibia. Note also the condition of the bones of the skull.

Treatment.—Light spinal support, to prevent any further fracture, with massage and exercises for the muscles of the back.

(IV) Fibrocystic Disease of Left Humerus showing Spontaneous Cure.

E. B., AGED 14 years.

This boy was first seen on March 8, 1920, when he gave a history of having fractured his left arm the year before. He had again fallen on the arm, and his mother was worried as to whether there was another fracture. X-rays showed no sign of fracture, but revealed a condition resembling either a myeloma or fibrocystic disease in the metaphysis of the left humerus. As the boy had no symptoms, it was considered unnecessary to operate, and accordingly watch was kept upon him. He was seen every few months, and has been under observation ever since. During this time, a series of skiagrams has been taken, from which one can see how the bone is gradually coming back to normal. As time goes on, the area of disease is moving further away from the epiphyseal line. This spontaneous cure supports the opinion that the condition is either traumatic in origin or, more probably, a defect in metabolism during the period of growth.

By ASTLEY V. CLARKE, M.D.

(I) Intense Ascites. ? Hepatic Syphilis.

BOY, aged 13 years, admitted to hospital March 27, 1928, suffering from intense ascites. Had been gradually ailing for eight weeks previously; stomach had been swelling for a week.

Family History.—Father and mother alive and well: five other living children.

Condition on Examination.—Very ill; severe ascites. Paracentesis: 15 pints of fluid withdrawn.

March 29, 1928.—Blood-urea 27 mgm.; urea concentration 2·79 per cent.; albuminuria; œdema of legs slight.

96 Clarke: *Intense Ascites; Tumour of Face*

Paracentesis required three times in less than three weeks; each time from 12 to 15 pints of fluid withdrawn. Nothing abnormal could be felt in the abdomen after withdrawal of the fluid.

The abdominal condition has improved and no further tapping has been necessary.

Treatment.—Ultra-violet light and mercury with increasing doses of potassium iodide.

No evidence of tuberculosis could be obtained, but investigation of the case shows that the kidneys were involved early in the history.

(II) Tumour of Face.

PATIENT, a boy aged 13 years. The growth began eighteen months ago and was at first rapid, then appeared to stop.

Since December, 1927, it has again been rapid.

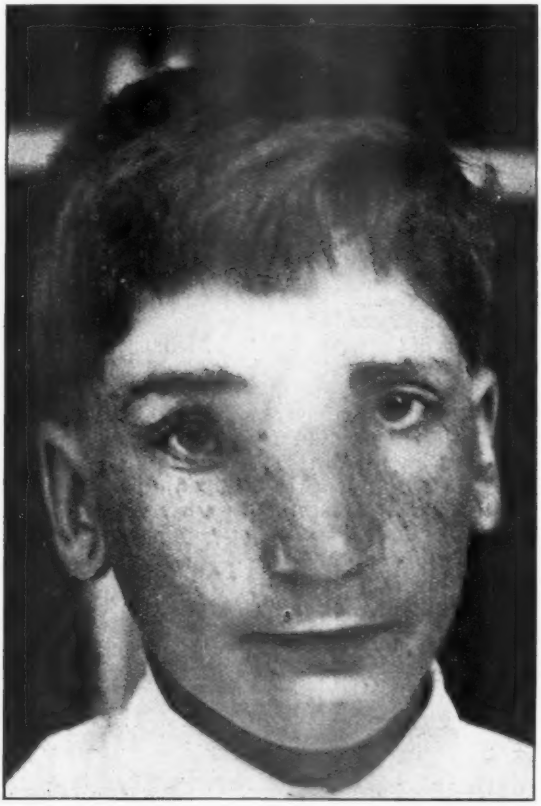


FIG. 1.

FIG. 2.

FIG. 3.

FIG. 4.

By J. D. SLIGHT, M.D.

Involuntary Movements of Arm. ? Psychoneurosis.

PATIENT, a girl, aged 15 years, has had involuntary movements of the right upper extremity for more than ten years. She is the oldest of the family, and at birth the mother had chloroform and delivery was by forceps. She has been left-handed from infancy. When she was two years old the middle-finger of her left hand was accidentally injured in a chaffcutter, and was consequently amputated. At the age of four she was sent to school, where the teacher found that she could not keep her right hand steady. No relative has been similarly affected. The movements have become gradually worse, especially during the last two years, and during the last few months have been noticed to extend to the face on several occasions. They cease during sleep, but sometimes prevent her from getting to sleep. They are worse on some days than on others, and are always worse towards night, and when in the presence of strangers. They interfere with the performance of voluntary

actions, but do not prevent it; she can touch her nose with her finger, and put her hand on her head, the limb shaking vigorously all the time. The right side of the face does not move so freely on speaking as the left, and the right palpebral fissure is slightly smaller than the left. There are no abnormal subjective sensations, and no clinical signs of change in sensibility. On plantar stimulation, the great toes move downwards or are stationary: in the latter case, the tensor fasciæ does not contract. There is no rigidity. Cerebration is not defective. The expression is not Parkinsonian. There are no visual or ocular signs.

By J. S. Sloane, F.R.C.S. (shown by Mr. Lawrence).

Case for Diagnosis. ? Fibrocystic Disease of Femur.

G. D. W., boy, aged 3 years.

March, 1926 (when 9 months old), pain in *left* knee and movements of left knee limited.

April, 1926.—Skiagram showed thickening outside lower end of *right* femur, apparently due to subperiosteal hæmorrhage.

Right Left

Fig. 1. (April 9, 1926).

November, 1926.—Skiagram showed what appeared to be cystic condition of lower part of right femur.

There have been no symptoms whatever in relation to the right knee; the condition was noticed only on X-ray examination.

The left knee has been somewhat enlarged and movement has been limited, and still remains so.

100 Sloane: *Case for Diagnosis;* Braithwaite: *Hypertelorism*

May, 1928.—Skiagram shows scattered calcified patches around lower end of left femur, and a filling in of the cavity in right femur, with a tendency to restoration of normal outline.

The left lower extremity is definitely longer than the right.

Left Right

FIG. 2. (March 4, 1928).

Extension and splints have been employed; at present a splint is worn only at night.

There is no "specific" history. General health has always been good.

This appears to be a case of "fibrocystic disease" arising from hæmorrhage into bone.

By J. V. C. BRAITHWAITE, M.D.

(I) Hypertelorism.

G. L., A GIRL, aged 2 years.

Backwardness noticed at age of 15 months.

Family History.—Parents healthy; one other child apparently normal.

Appearance.—Widely separated eyes, flat face, receding chin.

Measurements (November, 1925).—Distance between inner canthi 1·2 in.; between pupils 2 in; orbital height 1·25 in.; width 1·5 in.; index 83·3 in.; cephalic length 6·4 in.; breadth 5 in.; index 76·9 in.; circumference of head 18·75 in.

Vertical fissure down middle of frontal bone; hairy scalp showing a V-shaped peak in the mid-line.

Hypertelorism.

The blocks for these illustrations were kindly lent by the proprietors of the *Archives of Disease in Childhood*.

Anterior fontanelle was widely open (length 2·5 in., breadth 2·6 in). Ears large and stood from side of head; palate highly arched; eight teeth present; marked amyotonia; active rickets.

At age of 15 months could not stand, talk, or feed herself.

Put on pituitary extract.

February 11, 1927.—Teeth became carious.

May 3, 1927 (1 year 8 months).—Talking and standing.

November 27, 1927 (2 years 1 month).—Sang recognizable tunes.

March 6, 1928 (2 years 3 months).—Fontanelle closed.

(II) Cœliac Rickets.

C. K., FEMALE, aged 21 years.

Parents healthy; four other children living, eldest tuberculous; one died with rheumatic heart disease.

When 7 months old had diarrhœa, which lasted "on-and-off" for several years.

Legs gradually became deformed at the age of 3 or 4, when she began to walk. She has had no infectious diseases.

Came to out-patients department first on November 24, 1924, when she could hardly walk. X-ray showed marked deformity of both tibia and fibula, with flexion of the lower end of the right femur. The ends of the long bones appeared rather transparent.

The urine occasionally contained albumin.

Under massage and ultra-violet light she showed marked improvement in a month. She is still steadily improving and is working.

Has occasional headaches and attacks of nausea.

(III) Cœliac Rickets.

A. R., GIRL, aged 17 years.

The patient is said to have suffered from tubercular peritonitis at the age of 18 months; she had diarrhœa and vomiting and a large abdomen. She was ill "on-and-off" until she was 7 years old, when she improved considerably and began to walk. Before this she was undersized, but not deformed. After walking, deformities rapidly appeared in the legs.

For the last two years the patient has had attacks of tetany once or twice a week. Trousseau's and Chvostek's signs are positive. This is uninfluenced by calcium therapy and ultra-violet light.

Urine: No albumin, pus or casts.

(IV) Myositis Ossificans Progressiva.

B. O., A GIRL, aged 7 years.

Following fall at age of 2½ years, swelling commenced at back of neck; spread gradually to back and became hard. Lifting child caused pain.

On admission in 1924, both masseters, right trapezius, latissimus dorsi, rhomboidei and erector spinæ were swollen and tender. During the next few weeks, the muscles of the other side became affected with slight coincident pyrexia (99° F.). A portion of the right latissimus dorsi was removed for microscopical examination, and showed fibrosis and round-cell infiltration (figs. 1 and 2), but no ossification.

FIG. 1.—Myositis ossificans progressiva.

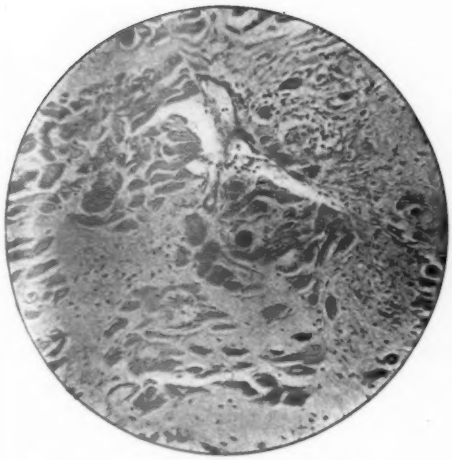


FIG. 2.—Section of muscle in stage of fibrosis. No ossification visible.

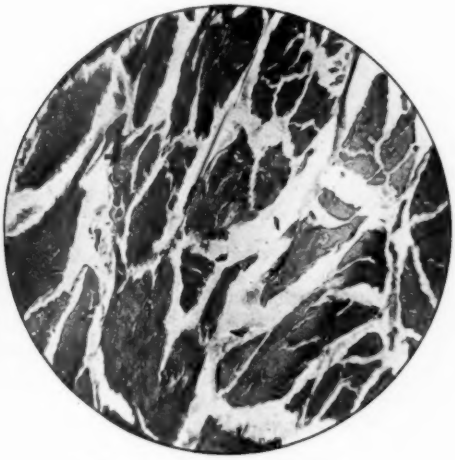


FIG. 3.—The same: high power.

104 Braithwaite : *Myositis Ossificans Progressiva*

Congenital deformity of hallux present. X-rays showed this to be due to presence of an extra digit in continuity with the head of the first metatarsal (fig. 3). X-rays showed no abnormality of affected muscles at this time, and the diagnosis of myositis fibrosa was made.

About six months later, ossification of the left pectoralis major was shown by X-rays; since then ossification has spread gradually to other muscles.

FIG. 4.—Radiogram of feet, showing extra digit in bony continuity with head of first metatarsal.

Electrical reactions (1924). All muscles responded to faradism, although a stronger current than normal was required to elicit contractions in the left trapezius and left pectoralis major.

Blood-count.—Hæmoglobin 62 per cent.; red cells 5,502,000; white cells 13,124 per c.mm. (polymorphonuclears 60 per cent., large lymphocytes 19 per cent., small lymphocytes 16 per cent., eosinophils 4 per cent., mast cells 1 per cent).

Wassermann reaction, negative.

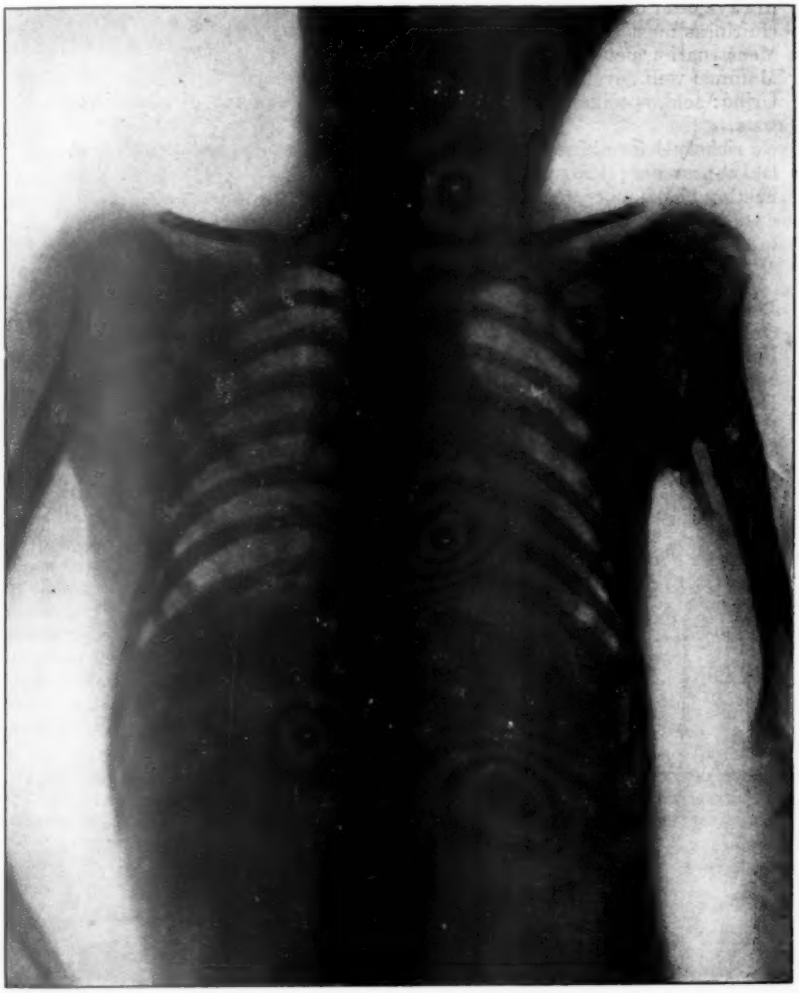


FIG. 5.

106 Braithwaite : *Infantilism and Hypertrichosis*

(V) Infantilism and Hypertrichosis.

O. J., A GIRL, aged 13 years ; height, 3 ft. 11 in.
Always been undersized and hairy.
Hairiness most marked in pubic region and axillæ.
Menstruation not yet commenced.
Mammæ well developed.
Urine : acid, specific gravity, 1015 ; considerable amount of albumin ; no blood ; no casts.
No rickety deformities.
Blood-pressure : 120 systolic, 25 diastolic.
Fasting blood-sugar, 0·1 per cent. Wassermann reaction, negative.

(VI) Osteogenesis Imperfecta.

J. J., GIRL, aged 2 years and 9 months.
Only child, parents healthy ; one cousin deaf mute.
First fracture (humerus) at age of 3 months.
Second fracture (tibia) at age of 4 months.
Had about ten more fractures in two years ; both femora, humeri, tibiæ.
Has not had a fracture during the past four months.
China-blue sclerotics : parietal bulging ; Wassermann reaction, negative Wassermann reactions of both parents negative.

Section of Surgery.

President—Mr. V. Warren Low, C.B., F.R.C.S.

[*February* 1, 1928.]

SPECIMENS.

Ruptured Kidney.

By Eric Pearce Gould, M.Ch.

This is the left kidney from a boy, aged 15, who fell, striking his left loin against the edge of a form. Immediate slight hæmaturia was followed three hours later by intense pain and swelling in the left loin, accompanied by progressive increase in the pulse-rate. At operation, four and a half hours after the accident, the lower third of the kidney was found detached, lying in a mass of blood-clot. The remainder of the kidney was removed. The patient recovered.

Carotid Body Tumour.

By J. E. H. Roberts, O.B.E., F.R.C.S.

The tumour was removed from a woman, aged 24, who had noticed the swelling for three years. Her tonsils had been removed four months previously, but the swelling, which was supposed to be due to a tuberculous gland, did not diminish in size.

At operation, the carotid artery was found to enter the tumour and its nature was recognized. The common carotid artery and its two terminal branches were ligatured and the tumour was removed.

Next day left hemiplegia developed, the right side of the face being also affected. There were mental symptoms of irrationality and irritability and semi-consciousness.

The mental symptoms passed away in about a week, and the facial paresis disappeared. The arm and leg began to recover in about a fortnight, and at the end of two months the leg had almost completely recovered.

Now, eighteen months later, the patient still has some spasticity and incoordination in the left hand, though the rest of the arm has almost completely recovered.

The report on the microscopical section is as follows:—

"The tumour is composed of alveoli of irregular size, containing collections of cells of the type normal to the carotid body separated by strands of fibrous tissue. It appears to be of the nature of a simple hypertrophy or adenoma. There are no signs of malignancy."

Carotid Body Tumour.

By C. E. Shattock, M.S.

Specimen removed from a woman, aged 45, in whose neck a swelling had been noticed for ten years. Her tonsils had been removed four years previously, as the mass was thought to be glandular.

The tumour was adherent to the trunk of the external carotid, which was removed with the mass.

Growth was very vascular, and microscopically resembled normal carotid body tissue.

96 Shattock—Riches—Ross

Branchial Cysts.

By C. E. SHATTOCK, M.S.

Three branchial cysts removed from right side of neck of a child, aged 5 years. The swelling had been noticed since the age of 2. The upper cyst lay just below the styloid process and was connected by a fibrous track with the two lower cysts, which were loosely adherent and situated at the level of the thyroid cartilage. All the cysts contain sebaceous matter and are lined by stratified epithelium.

An almost complete ring of hyaline cartilage is present in the wall of the upper cyst.

Ruptured Ovarian Pregnancy.

By E. W. RICHES, F.R.C.S..

Clinical History.—Aged 34; one child, aged 4 years.

Periods regular, slight blood-stained discharge four months ago, repeated after each subsequent period.

Severe hypogastric pain five days before admission, repeated on the day of admission and felt in the right supraspinous region and back of the neck on lying down.

Admitted to the Middlesex Hospital, under Mr. A. E. Webb-Johnson, on January 15, 1928, with signs of intra-abdominal bleeding and severe pain in right shoulder on lying down.

Laparotomy: Masses of blood-clot and blood-stained fluid in the pelvis; both tubes normal; uterus enlarged to size of a tennis ball.

Left ovary had a dependent plum-coloured swelling the size of a walnut, from which bleeding was occurring. Left ovary removed.

Specimen.—Left ovary with attached pregnancy; no sign of a fœtus.

Ovary shows large corpus luteum of pregnancy.

Sections of plum-coloured mass show well-marked chorionic tissue.

Two Brain Tumours.

By J. P. ROSS, F.R.C.S.

(I) A LARGE degenerating glioma in the left frontal region, invading the corpus callosum, and also impeding the flow of cerebro-spinal fluid, causing internal hydrocephalus. The anterior horns of the lateral ventricles were obliterated, but the posterior portions were dilated.

The interest of the specimen lies in the fact that there was no clinical evidence of frontal lobe lesion; speech, memory and attention were perfect, and behaviour was normal; and there was no weakness of any part of the body—and this in spite of the great size of the tumour. On account of severe headache of short duration, intense bilateral symmetrical papillœdema, and slight ataxia and atonia of the left arm, the tumour was diagnosed as a left cerebellar glioma.

(II) A large glioma, very soft, deep to the insula on the right side and extending to the base of the brain, infiltrating the right olfactory tract, and also causing some dilatation of the left lateral ventricle owing to cerebro-spinal fluid block.

A tumour of the cerebellum was suspected, as the patient showed well-marked nystagmus, and was very giddy, weak and ataxic, especially on the left side. The diagnosis was not clear, and a ventriculogram was made which showed a blockage of the right foramen of Munro. This localized the tumour correctly. Death occurred suddenly after a series of fits, three weeks after an exploratory and decompressive operation had been carried out.

Specimens from a Case of Spontaneous Fractures.

By W. D. DOHERTY, M.Ch.

THE specimen shown is from a patient, Miss H., aged 52, who was originally admitted to the hospital on April 18, 1927, with a simple fracture of the left femur in the lower third. X-rays showed no obvious disease in the bone; she was treated in extension and was discharged from hospital on August 7, 1927, with firm union of the left femur, although a rather large mass of callus had formed at the site of fracture.

Three weeks after discharge from hospital she fell and fractured her right leg in several places, the tibia and fibula in the middle of the shaft and the femur in three places. She was treated at a local hospital in plaster, but came under our care again on January 1, 1928, as there was no sign of union in the tibia or femur and there was considerable deformity. Radiographs taken at this time show a diffuse growth involving the right femur and tibia, and also a large mass of growth at the site of the old fracture in the left femur. Skiagrams of other bones reveal a deposit in the lower end of the right radius.

There is no evidence of primary malignant disease in other parts of the body. The urine contains no abnormal constituents. Owing to the severe pain in the ununited fractures, and at the urgent desire of the patient, the right leg was amputated through the upper third of the femur on January 17, 1928.

The specimen exhibited shows the right femur and tibia cut longitudinally. There is a diffuse reddish growth throughout the shaft of the femur with cystic cavities in places. In the region of the fractures the growth has spread beyond the compact bone, but is everywhere limited by the periosteum. There is a large deposit of the same type of growth in the middle of the shaft of the tibia. Here the growth appears to be of much firmer consistency, and there is a tendency to bone formation to be felt in it. The growth itself has the appearance of a myeloma, and the microscopical section reveals a large number of multinucleated giant cells with a ground-work of fibrous tissue and spindle cells.

Invasion of Bone by Periosteal Sarcoma.

By JULIAN TAYLOR, M.S.

IT is a remarkable thing that even at the present time there is no clear and generally held conception of the radiographic appearances of malignant tumours invading bone, except with regard to certain well-known signs such as the radial striæ that are held to characterize the radiograms of certain kinds of growth. What I have to put before you is an attempt to correlate the morbid anatomy with the radiographic appearances of a typical example of periosteal sarcoma.

Scheme of the Examination.—A thin slice of the whole length of the affected bone, cut with a band saw, was radiographed, and at selected levels histological examinations were carried out.

The example demonstrated is a tibia amputated from a boy, aged 16, the seat of a very rapidly-growing periosteal sarcoma that began apparently at the site of slight repeated injuries received on the same spot on his leg during the pursuit of his occupation. The tumour caused great pain and was associated with fever.

The radiographic appearances of the slice of this growth show that a large amount of new bone has been deposited. The outline of the clinically palpable tumour at the upper extremity of the tibia is distinctly seen, and there is some destruction of the cortex where the tumour is in contact with it. New bone has been deposited, of different architecture in the different parts of the bone:—

98 Julian Taylor: *Invasion of Bone by Periosteal Sarcoma*

(1) In the tumour itself are the thin radial striæ that are regarded as characteristic of this type of growth, but that are also seen in other kinds of malignant invasion of bone.

(2) In the cancellous bone and medullary spaces underlying the main mass of the growth there are a patchy destruction and an intense condensation of the bony tissue that extend for some distance distally to the main mass.

(3) There is a thickening of the cortex that extends as far as the junction of the middle and lower thirds of the tibia. This thickening is seen on both the superficial and deep aspects of the cortex and has the characters of normal bone. On the surface the new bone is seen as a subperiosteal deposit laid down parallel to the shaft (i.e., as laminæ concentric with the cortex), in the manner usually seen in inflammatory deposits. The thickening of the cortex increases proximally until, at the edge of the main mass of the growth, the newly-deposited cortical bone gives place to the radial striæ of the tumour already described.

No one will question that these are common radiographic appearances in periosteal sarcoma, but as evidence that this is so, I show an essentially similar picture of an example affecting the femur of a boy.

Microscopical examinations of the various regions show the following appearances:—

The growth is a mixed-celled sarcoma, but whereas in the main mass of the growth the cells are mostly spindles, the outlying infiltration is of a less highly differentiated type.

Just above the malleolus the surface of the cortex is normal, but the subperiosteal layer of osteoblasts shows a slight increase of activity. A little above this point and below the visible new bone of the radiograph, there has been deposited a layer of new bone that is fused with the cortex. There is greatly increased subperiosteal activity, the osteoblasts being swollen and numerous.

More proximally there are concentric laminæ seen in section, and in the vascular spaces between them there are tumour cells surrounding vessels.

A higher magnification of the subperiosteal tissue shows great activity, with here and there tumour cells among the proliferating osteoblasts.

Still more proximally there are well-formed concentric laminæ and, again, vascular spaces containing tumour cells alongside vessels.

Just distal to the edge of the main mass the early formation of radial elements is seen, while in the tumour itself there are seen long thin strips of radially arranged bone, whose osteoblasts are easily distinguished from the spindle-celled growth alongside. Destruction of bone is also seen to be occurring.

It is thus clear that in this example there is a widespread invasion of the medulla and, especially, of the cortex, alongside the vessels of which the infiltration seems principally to occur. This invasion has resulted in considerable new bone formation, which, apart from the fact that it is invaded by tumour cells, has normal characters. In the main mass of the tumour the new bone is laid down in radial striæ.

The clinical importance of these findings is first, the extent of the invasion of the bone and secondly, the fact that were it not for the visible outline of the growth, and the radial striation within it, there would be no radiographic certainty that the disease was sarcoma and not an inflammation. I show a radiogram of an early case of periosteal sarcoma of the lower end of the femur, in which no new bone has been formed in the tumour; the outline of the growth is indefinite; there is practically no destruction of the underlying cortex; and a thin slip of new subperiosteal bone, of the type usually said to be inflammatory, was the only radiographic sign that enabled a diagnosis to be made.

Some Cystic Appearances in Bone.

By J. MAGNUS REDDING, F.R.C.S.

RADIOGRAPHIC RECOGNITION.

THE radiographic diagnosis of simple cyst of bone depends on the recognition of (1) a more or less regular and definitely circumscribed area of comparative loss of bone structure (not complete loss, since the walls of the cyst are superimposed over the central area of complete loss); (2) the fact that the margins of this area of translucency are well defined in every part, and may show very slight linear condensation; (3) the absence of any surrounding alteration in bone structure.

The definite margins and abrupt transition to normal bone structure, if these features can be convincingly demonstrated, are sufficient to exclude acute inflammatory diseases and malignant neoplasms; primary central sarcoma rarely causes any serious difficulty, but secondary malignant deposits may require very careful examination to avoid error.

The absence of any abnormality in structure around the cyst excludes the majority of chronic inflammatory lesions; occasionally a small central abscess cavity may present appearances hardly distinguishable from cyst, except for the small size of the lesion when first seen—a point to which reference will be made later. Before leaving the subject of the radiographic recognition of these central cystic conditions, I would like to point out that surgical interference completely destroys the chain of evidence whereby a diagnosis is reached.

Site of Bone Cysts in Order of Frequency.—Much the most common site in my experience is (1) the upper third of the diaphysis of the humerus in young children.

Next in frequency are: (2) The scaphoid in adults. (3) The lower end of the femur and upper end of the tibia in adults. (4) The upper end of femur in adolescents. (5) The extremities of the fore-arm bones.

All are rare except (1) and (2).

SCOPE OF X-RAY DIAGNOSIS.

I have purposely avoided a precise name for these cystic conditions, because the radiographic appearances are characteristic, not of any single pathological entity, but of the group which may be called the benign central tumours of bone; in other words, these radiographic cysts may be found to contain fluid, fibrous connective tissue, myelomatous tissue or cartilage, or a mixture of, at any rate, the first three. Radiographically, one can recognize a benign central tumour with considerable certainty—the accuracy of diagnosis in this respect will bear favourable comparison with histological examination; but beyond the recognition that the lesion belongs to this group it is frequently impossible to go.

Central chondromata, fluid cysts and fibrous tissue cysts are quite indistinguishable one from another; all have a tendency to extend in the long axis of the shaft without causing much expansion. Myelomatous tumours tend to produce considerable expansion, so that the central cavity may become almost spherical. Many instances are met with, however, in which the appearances are not sufficiently typical to justify any expression of opinion on the contents of the bone cyst. The site of the cyst, again, and the presence or absence of so-called "trabeculation" are factors which will often be found misleading.

100 Redding: *Some Cystic Appearances in Bone;* Veau: *Hare-Lip*

Pathology of Central Benign Tumours.

If central chondromata be excluded, no explanation of the pathology of these central benign tumours has yet been put forward which can command general acceptance. I would like merely to record two personal observations which, if they are sustained by the experience of others, may be of some interest in this connexion.

(1) The common site of occurrence, clinical features, radiographic appearances and results of operation are in essentials fairly constant, and are, to some considerable extent, independent of the contents of the cyst; while in some of the solid central tumours, different portions of the tumours may present respectively the histological characteristics of myeloma and of fibrous connective-tissue tumours; examples are also on record where a change from one of these characteristics to another has occurred, as shown by portions removed at different times from the same tumour. It seems possible, therefore, that some single, underlying primary pathology may account for all these conditions, and that differentiation into myelomatous or fibrous tissue or fluid may depend on some secondary factor.

(2) If one takes the most common of these cysts, that in the upper end of the humerus, it is remarkable that it is never seen until of a size sufficient to cause symptoms, although its subsequent increase in size is slow. In hospital practice one sees very many skiagrams of the upper end of the humerus of young children, chiefly taken because of suspected fracture of the clavicle, and it appears strange that one has never once seen an unsuspected cyst. I have wondered, therefore, if these cysts in the humerus do not make their first appearance as lesions of considerable size, owing to a fairly rapid transformation of structure affecting every part of a comparatively large area of bone simultaneously, brought about, perhaps, by some localized nutritional, metabolic, or developmental variation from the normal.

Treatment.—At the present time there appears to be a good deal of support for the conservative treatment of these cystic conditions when, as frequently happens, they are first recognized as the result of a more or less spontaneous fracture. Fracture through the cyst commonly results in extensive calcification in the central area, sometimes followed by definite ossification and complete obliteration of the lesion. In many cases, however, temporary diminution in the size of the cyst is followed, after a lapse of some months, by recurrence. It would appear reasonable to suppose that the success of conservative treatment depends largely on the nature of the cyst contents, which, as emphasized above, can rarely be predicted with certainty from the radiographic appearances.

[*June* 6, 1928.]

DISCUSSION ON THE TREATMENT OF HARE-LIP.

Professor Victor Veau (Paris).

During the past thirty years I have performed more than one thousand operations upon hare-lips, and have seen and studied over one hundred patients operated upon by other surgeons.

I have made many mistakes, and have tried to discover why I did not obtain the results that I expected, and I have learnt that the surgeon must aim at reconstructing the layers in the exact position in which nature has placed them.

As the foundation of this surgery, I have studied the part the median tubercule plays in the constitution of the face. I have deduced a scheme which is my guide in the method which I indicate.

I shall discuss only the unilateral total hare-lip, which is the most frequent[1] and the most schematic.

Unilateral total hare-lip is characterized by the cleft of the alveolar border. There are typical cases and varieties; the typical cases are 72 per cent. of the total of unilateral total hare-lip. Figs. 2, 3, 4, 5 represent that form, fig. 2 is of a child of three weeks, fig. 3 of seven weeks, fig. 4 of two years, and fig. 5 of a girl of twelve.

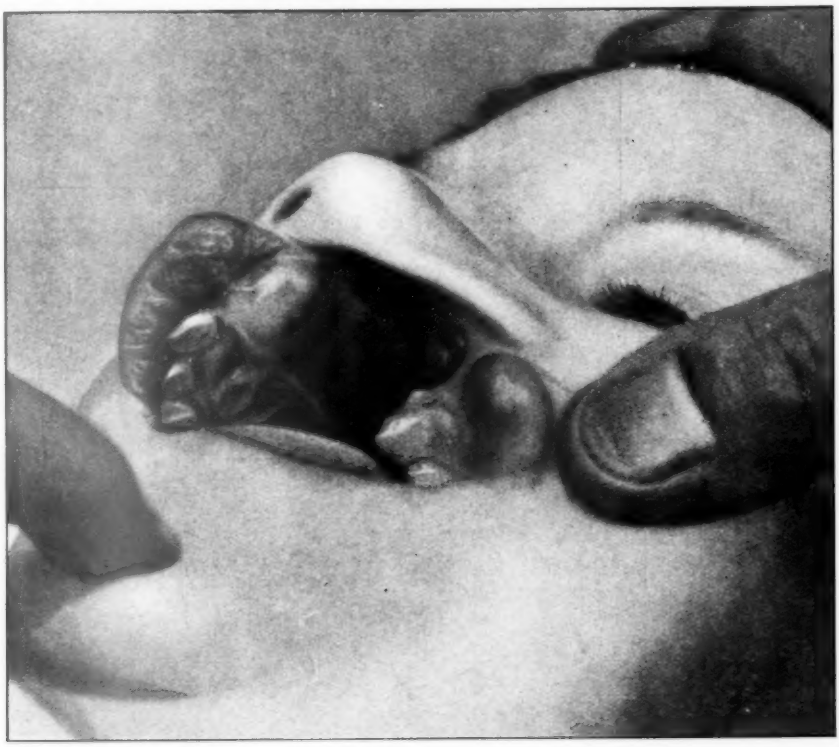


FIG. 1.—Unilateral total hare-lip. A child of eighteen months.

There is a fundamental difference between the walls of the cleft; they are formed on each side by the skeleton and the lip.

The internal wall is extremely prominent owing to the protrusion of the intermaxillary with the incisors. The bone projects forward, more alarmingly than

1 In an article on "les formes anatomiques du bec de lièvre ordinaire" (*Ann. d'anat. Patholog.*, May, 1925) I have indicated the relative frequency of the different forms, from 400 patients, whom I have observed, before any operation.

Unilateral simple	...	...	...	33 per cent.
Unilateral total	...	...	...	48 per cent.
Bilateral simple	...	...	...	7 per cent.
Bilateral total ...	...	...	...	12 per cent.

FIG. 2.

FIG. 3.

FIG. 4.

FIG. 5.

FIGS. 2, 3, 4, 5.—Typical forms of unilateral total hare-lip.

dangerously. Before the cutting of the teeth at the age of our operations, this bone is covered by a hard, adherent mucous membrane, which cannot be utilized for the plastic. When the suture of the lip is finished, the protrusion of the bone is spontaneously reduced. The lip on this internal wall is drawn flat; it is necessary to suture the deep muscles in order to produce a good lip.

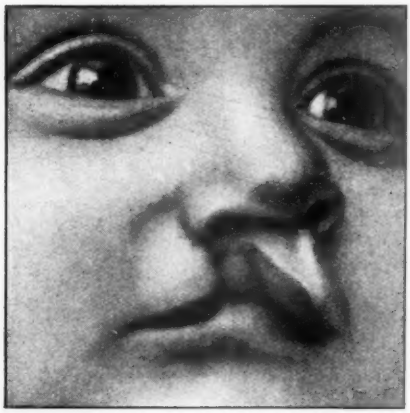


FIG. 6.

FIG. 7.

FIG. 8.

FIGS. 6, 7, 8.—Unilateral total hare-lip variety with bridge.

The external wall, on the contrary, remains behind; it is gathered together, but it is like india-rubber, it encloses powerful muscles of marvellous elasticity. As soon as the frenum attaching the lip to the depths is sectioned (fig. 1), it can be brought inside like a curtain in front of the hole which it closes. This frenum is

constituted by the myrtiform muscles. In a recent work I have discussed the details of this frenum, which is the key of the operation.

The bone of this outside edge is very deeply placed behind the protrusion of the premaxilla and is not at all adherent to the soft part of the lip; this makes the operation much easier.

I have shown that all the floor of the nostril belongs to the median tubercle. To make a normal nostril you must take this floor on the inside wall; to do that I detach the mucous membrane widely from the premaxilla and the septum, and I suture it at the outside edge, which has been liberated so as to be mobilized in front of the cleft. This form represents 72 per cent.

Anatomical Varieties.—Besides these typical cases there are two varieties which the surgeon must know.

FIG. 9.—Unilateral total hare-lip without cleft palate.

(1) A bridge of integument is stretched between both walls (figs. 6 and 7).

These cases are much more favourable for the operation, because it is relatively easy to obtain a normal nostril. We shall see that the spreading out of the nostril is the great stumbling-block to the treatment of these cases.

This bridge is always in the same place, but it varies very much in thickness. Fig. 8 represents a filiform bridge; the photograph was taken soon after birth. This bridge broke spontaneously some weeks after.

These cases form 18 per cent. of the total of unilateral total hare-lip.

(2) *Without Cleft Palate.*—In 11 per cent. of the cases the cleft is localized at the lip and at the alveolar border. Fig. 9 represents this anatomical state. These cases are difficult to treat; the total disunion of the lip is more frequent than in typical cases.

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Principles of Operation.

In the treatment of unilateral total hare-lip, I try to avoid two defects almost always present in children correctly operated upon by the classical method.

(A) Spreading of the upper lip.

(B) Flattening of the nostril.

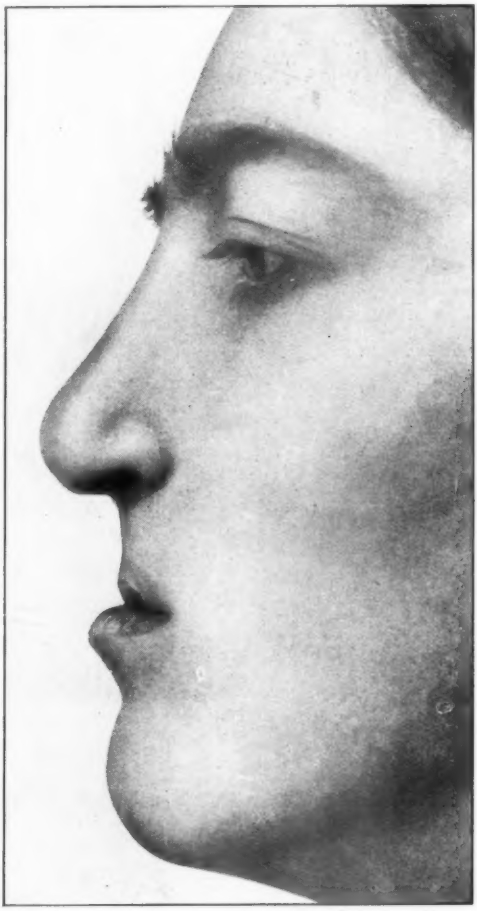

Fig. 10.—Flattening of the upper lip.

(A) The spreading of the upper lip is shown in fig. 10, taken from a child operated upon by Dr. Ahrmann (of Mulhouse). When the lower lip projects more than the upper the profile is very ugly. A frequent cause of spreading out the upper lip is the section of the bone done by some surgeons to push back the premaxilla, as they think this protrusion sometimes makes disunion noticeable.

This is a regrettable practice—an error and a fault. The pushing back of the premaxilla does not prevent the nostril from spreading, gives an ugly lip, and greatly disturbs the development of the teeth. I absolutely condemn the method. Numerous photographs prove that I have succeeded in giving normal protrusion to the upper lip. I have almost always succeeded by two practices :—

(1) I carefully suture the muscles of the lip, because I maintain that the protrusion of the lip is produced solely by normal growth of the muscles on a bone sufficiently projected.

(2) I do not take away the tegument, I preserve it wholly—but this is much less important than the suture of the muscles. Flattening of the nostril: figs. 11 and 12, taken from a boy operated upon at Leipsic, illustrate this defect.

I have not always succeeded in completely avoiding it, but I now obtain nostrils that are not so flattened as formerly.

FIG. 11.

FIG. 12.

FIGS. 11, 12.—Spreading out of the nostril.

I carefully reconstruct the floor of the nostril with a big flap of mucous membrane from the septum.

Fig. 13 shows that in the young child at the age when we operate, the vomer is always curved. I take the mucous membrane of this curved septum and turn it to suture it to the external wall of the cleft. This method is derived from embryological conditions which I have discerned elsewhere. Normally, all the floor of the nostril is formed by the premaxilla. This premaxilla has remained inside the cleft of which it is rational to take the floor in order to replace it in its right position.

Happily the mucous membrane of the septum has marvellous plastic qualities; it is tough, never breaks, can be detached very easily and never mortifies, because it is very vascular.

This practice has moreover the advantage of beginning from the first intervention the reconstitution of the palate, which becomes very easy.

There is a difficulty in the detachment of the big flap of mucous membrane of the septum, which is at the level of the furrow, behind the premaxilla; it is useful to begin the detachment very high on the septum, and to proceed from top to bottom. If the difficulty is appreciated, this is easily done, but great care must be taken at this point.

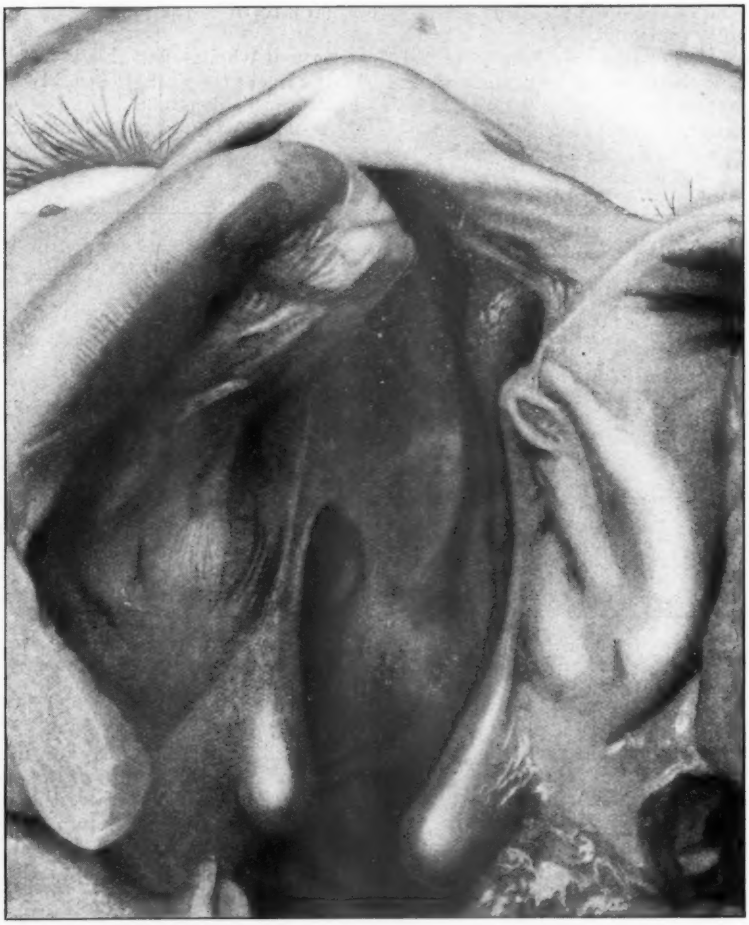

FIG. 13.—Palatine surface of unilateral total hare-lip.

Both spreading of the upper lip and flattening of the nostril are defects peculiar to the total form of the unilateral hare-lip. There are others common to both forms. The most frequent is the irregularity of the cutaneo-mucous line. A lip can only be well formed if the continuation of this line is perfect. There must be neither the

enclosing of mucous membrane in the skin (a frequent defect) nor the enclosing of the skin in the mucous membrane (a rare defect).

The lip is better when this cutaneous mucous line is very apparent, as its projection catches the light.

I have succeeded in making this line very apparent in tracing the little flap which is represented in fig. 17.

The ideal lip is known in France by the English name of Cupid's bow (fig. 23).

I do not know to what anatomical cause this curve is due. I obtain it sometimes, by the technique represented, but not always.

I do not know to what cause this difference in results is due, but I am still trying to discover it and do not despair of succeeding, by a slight modification of technique, in producing this curve, which makes the lip perfect.

Technique.

I now operate as follows:

First step: reconstruction of the floor of the nostril.

(*a*) A flap of mucous membrane is taken from the septum and turned outwards. The incision begins at point A (fig. 14) on the side of separation (very easy to

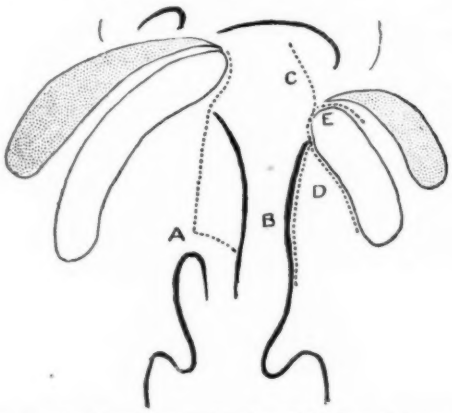

Fig. 14.—Incision for reconstruction of the floor of the nostril.

recognize) between the white buccal membrane and the red nasal mucous membrane. The incision goes forward and turns round the premaxilla to the insertion of the lip (fig. 14).

Another incision is made across the vomer, perpendicular to the first and starting from A (fig. 14) to permit the turning over of the flap. This flap of nasal mucous membrane is easily detached and is a marvellous flap. It is difficult only on the anterior part, on the level of the premaxilla and in the ridge behind the premaxilla. The detachment is easier at the upper part of the septum. The unstuck flap is turned over (fig. 15). On account of its elasticity it can be carried very far; however large the cleft may be, it reaches and goes beyond its external edge without difficulty.

(*b*) Incision of the external wall.—This incision is more complicated in appearance than in reality (fig. 14). It begins along the palatine edge (fig. 14 B) and continues in the nostril (fig. 14 C), remaining close to the bone.

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An incision is made across the ridge between the lip and the gum (fig. 14 E). Finally, a fourth incision is made along the external edge of the palatine plate.

Then the palatine mucous membrane is completely detached. This is very easy because it is not at all adherent. This detachment is extended as far as the posterior palatine artery, which is always preserved.

The detachment of the ala of the nose is very important; it permits the outside wall to be brought into contact with the inside wall. It is very easy as it is not at all adherent to the bone.

The region is then prepared for the suture (fig. 15). Behind, the palatine mucous membrane is sutured on the vomerian flap. In front, the edge of the flap is sutured at the cutting of the nostril.

Second step: reconstruction of the lip.

(a) Incision of the internal wall of the lip.—I cut the flaps as indicated by figure 16, taking great care that the line of incision is completely in the skin or in the

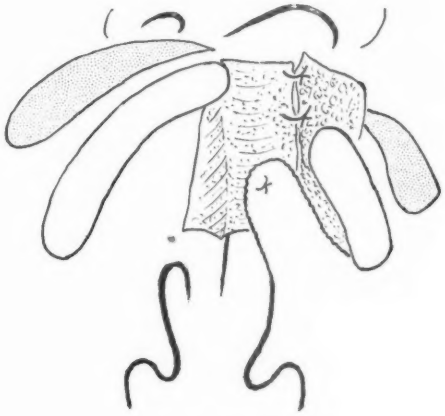


FIG. 15.—Suture of the flap.

mucous membrane; it is very important for the definitive scar. There must be no reciprocal enclosing.

The edges of incision are separated and quite isolated, the orbicular muscles are seen in the deep parts of the field.

The arrow (fig. 16) indicates the direction where the detachment must be carried very high on the septum. The higher you go the more you lengthen the flap which must constitute the floor of the nostril.

(b) Incision of the external wall of the lip (fig. 17).—The end of the incision rejoins the incisions already made.

Difficulty arises from the fact that the lip is very variable, and also that as soon as you have begun the incision the field is obscured by blood. It is necessary to know exactly what you have to do, or else you are lost, and the result is a bad scar.

The separation of the line of incision must be carried rather far—it is necessary to see the muscles.

(c) Deep sutures.—I then place two stitches which I consider very important; thanks to them, total disunion is never observed, and partial disunion is very rare.

The first stitch is for the ala of the nose—outside, it is passed as shown in figure 18, it grasps the muscles which are inserted on the ala. Inside, it is passed as also shown in figure 18. Both threads are knotted on a tiny piece of folded gauze. I have very

FIG. 16.—Incision of internal wall.

FIG. 17.—Incision of external wall.

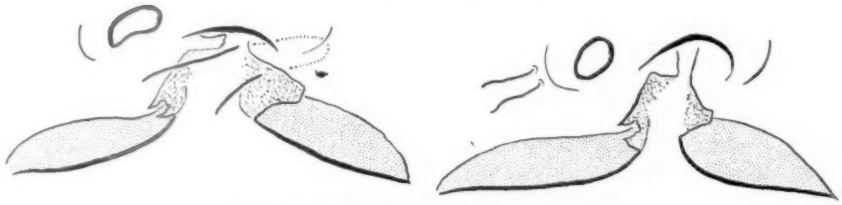


FIG. 18.—Deep stitches for the ala of the nose.

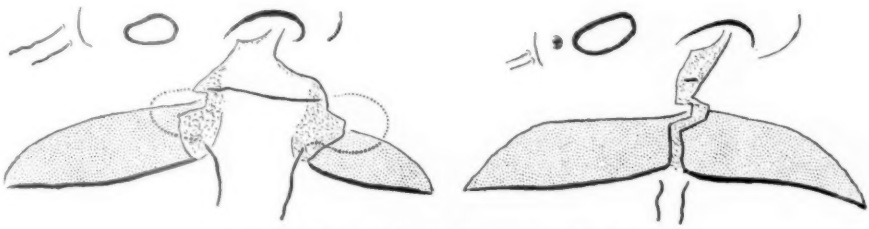


FIG. 19.—Deep stitches for the muscles of the lip.

much varied my manner of placing these stitches. What I represent here is what I do now—perhaps it is possible to do something better.

The second stitch is for the muscles of the lip (fig. 19).

It is passed through the middle of the muscles and is knotted outside, taking any segment of mucous membrane in its loop. This thread is difficult to take out, but

FIG. 20.

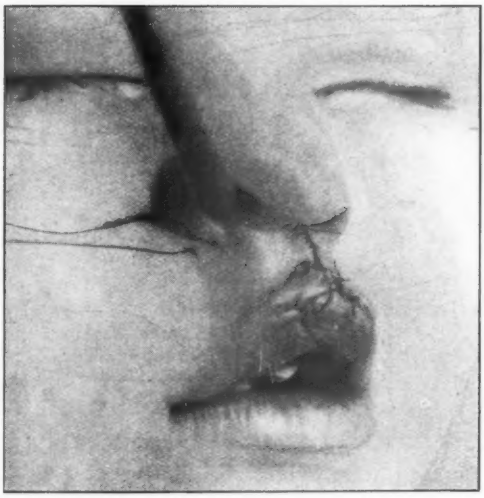


FIG. 21.

FIGS. 20, 21.—Unilateral total hare-lip, before and immediately after the operation.

if you do not include any segment of mucous membrane you have less risk of infection.

(d) Suture of the skin.—The suture of integument must be very carefully done.

FIG. 22.

FIG. 23.

I use a very fine straight needle, threaded with arterial silk. To be sure of having a very good co-adaptation I use magnifying glasses arranged as spectacles.

The suture is prolonged into the nostril, as high as possible.

FIG. 24.

FIG. 25.

FIGS. 22, 23, 24, 25.—Unilateral total hare-lip, before and after operation.

Results of this technique are shown in the figures from 20 to 25.

I wish to point out that my photographs, after operation, have not been retouched, and that the subjects were not "made up" with rouge or powder.

In cases of hare-lip there are always enough tissues, whatever the size of the cleft, to reconstitute a normal lip. It is for us to perform an anatomical operation which corrects the malformation by reconstructing all the layers which are divided.

Mr. C. H. FAGGE

said that for purposes of description hare-lips could be divided into three classes: (*a*) partial, (*b*) unilateral, usually associated with cleft palate, (*c*) bilateral. This subdivision was one of degree only, for, in all three classes, similar defects were present and similar problems had to be dealt with. In his experience class (*a*) was very rare and was usually associated with defects of the ala, so that it was necessary to plan operation on the same lines as that for a complete hare-lip, carrying the incisions into the nostrils, and freeing the ala, so as to bring it towards the middle line. In his experience operation on the lip alone was usually inadequate.

His views on the operative methods that should be adopted were based on the fact that, in his opinion, the tissues were deficient in every case, so that if the lip was made of normal depth, it would be defective in width and vice versa. The first defect which had to be dealt with was splaying out of the ala. This was corrected by mobilizing the ala and soft tissues of the cheek as far as the infra-orbital ridge. When bringing the ala inwards it was usually necessary to cut away part of the circumference of the nostril; this was done at the inner margin of the cleft: the reason for this was that otherwise the nostril might be too wide, and a wide nostril was second only to a deficient or rigid soft palate in the causation of defective speech; at the same time the nostril must on no account be made unduly narrow, for it was essential that natural growth should restore the symmetry of the face, which operation only made possible, and naturally growth could take place only if normal respiration was established through the nostril of the affected side. If the ala nasi was brought into the correct position the naso-labial sulcus would be restored, but there were two defects for which he (the speaker) had so far found no adequate remedy: the first was the eversion of the ala. He had tried several methods in order to convert this into the normal inverted ala, but with only a very partial success. The second was depression of the apex of the nose on the affected side. [Photographs illustrating this deformity were shown, and it was pointed out that some of these showed that the process of growth tended to diminish the deformity.]

His own lip operation was founded on that of Mirault, but the incisions did not include the mucosa of the inner surface of the lip; the flap produced on either side was turned backwards and the two margins sutured together after excising what was redundant; this was done to prevent the lip becoming adherent to the alveolus. It was important to insure that no mucous surface was left at the angle of the larger fragment, and the flap brought from the lateral margin of the defect to form the tubercle of the lip should be larger than appeared necessary at the time as shrinkage took place.

A very large number of his cases had been operated on during the first month or two of life when the lip operation was performed at the same time as that on the soft palate. The flaps of mucous membrane turned backwards towards the mouth were sutured with fine catgut and the muscles and connective tissue on either side were brought together by a continuous catgut stitch, the skin and lip edge being united with interrupted stitches of gossamer gut.

In some cases, particularly those of double hare-lip, a secondary operation was necessary to bring the upper lip forward, so as to correspond with the lower lip. He had never removed the premaxilla when operating on a double hare-lip, and many years ago gave up dividing its vomerine stalk, but in spite of this such patients were often "underhung," and this, in his opinion, was chiefly due to the tightness of the tissues of the lip. This deformity could be overcome by means of a prosthetic apparatus which carried any necessary teeth.

At the second operation the lip was freed from the alveolus and the resulting raw surface epithelialized by means of a Thiersch graft after the method of Esser; the graft was secured in position by a mould of dental stent. By this means the superior labial fornix was deepened and Mr. Kelsey Fry was able to fit a suitable prosthetic apparatus bringing forward the upper lip and in most cases completely overcoming the deformity.

Mr. T. Pomfret Kilner.

In Mr. Gillies's absence I am called upon to contribute to this discussion something from our joint experience.

The programme left little doubt that primary closure of lip clefts would be very fully discussed by earlier speakers and I therefore decided to deal chiefly with secondary operations on cases in which the primary operations have left much to be desired. Mr. Gillies and myself have had a considerable number of such cases referred to us during the last few years.

Before describing these operations, however, I should like to put forward a plea for perfectly simple primary reconstruction, point out those principles which we think should govern all hare-lip work, and briefly outline our own technique.

However small the parts, each element of the lip possesses its separate and distinct layers, and these, particularly the muscle layers, must be correctly approximated if balanced function is to result.

As in all plastic work, raw surface must be reduced to an absolute minimum, for raw surface means scar tissue and contraction.

We feel that no surgeon is justified in promising a perfect cosmetic result in one operation. The establishment of function, which leads to natural development and produces in the lip an ideal state for later cosmetic procedures, should be his main object.

All zig-zag incisions and flaps are, in our opinion, to be condemned. Our practice is to concentrate on the nostril and worry very little about the vermilion margin. We never try to equalize the sides of a cleft by elongating the shorter of the two elements by a Rose-type skin incision, for we feel that, if anything, the longer of the two sides requires shortening for the production of a good cosmetic result.

No skin is sacrificed in the operation, and only that small amount of mucous membrane on the border of the cleft which has probably been stretched round from the back surface by muscle pull and is usually thin, poor material, is sacrificed: even this is not removed but is reflected backwards as a flap, until it has been seen that there is ample material to approximate on the back surface of the lip.

It is our custom, in unilateral clefts, to free the smaller element considerably from the maxilla and to anchor the deep tissues of the alar base to any deep tissue available in the region of the nasal spine.

Mr. Gillies, in these cases, frequently turns over a small flap of muco-periosteum from the stump of the alveolar process on the longer side. This small flap comes to lie with its raw surface in apposition to the raw surface of the advanced alar base when the suturing is completed, and so helps materially in preventing the backward contraction of the ala which is so frequently seen.

I have been much impressed by the work of Ritchie, of Minnesota; he considers the alveolar cleft the key to the treatment of the lip cleft and, when the gap in the alveolus is at all wide (more than 6 mm.), carries out a preliminary submucous wiring to approximate the alveolar stumps before reconstructing the lip. When the alveolar gap is narrow, the lip closure in itself may be relied upon to bring about

this approximation more gradually. Such wiring must not be confused with that of the Brophy cleft palate operation: it aims, not at a bodily approximation of the maxillæ, but at a general easy moulding of the alveolar arch. It appeals to me as a treatment correct in principle and tending to produce better contour and more regular dentition. Further, it cannot help but simplify the closure of the lip cleft and cut down the necessity for lateral freeing of the elements to a minimum.

Ritchie also goes to considerable trouble to obtain accurate muscle approximation, using electrical stimulation to ascertain the precise upper and lower limits of the muscle in each element.

Though I have not yet had an opportunity of properly trying out these two procedures, I feel justified in mentioning them because of their essential soundness and because I think that the preliminary moulding of the alveolar processes will ultimately replace Gillies's muco-periosteal flap.

In bilateral clefts with protuding premaxilla we carry out the usual premaxillary replacement at the time of closing the lip clefts, but here again I feel that greater success awaits us when we adopt Ritchie's method of bringing back the premaxilla more gradually by wire loops, tightened periodically, before attempting the lip closure.

We never quite know how far the prolabial skin belongs to the lip and how far to the columella. We tend to place it higher and higher in the lip reconstruction, for, in secondary operations, we so frequently have to take it out of the lip altogether, before the nose tip can pass forward into normal position. Wherever it is placed, it is our practice to join to its deep tissues the muscle tissue of the upper parts of the lateral elements, approximating the lower fibres of the un-united orbicularis below the point of the "V."

We use deep catgut stitches for muscle approximation; catgut for the mucous membrane on the back of the lip; and interrupted sutures of the finest ophthalmic silkworm gut, mounted on eyeless needles, for skin and vermilion approximation.

To prevent tension, we use Logan's traction bow, a simple instrument not sufficiently known in this country. When properly fixed it takes all tension off the suture lines and yet leaves the operation area open for inspection, regular cleansing and later removal of stitches. It should be kept in position for about ten days if early stretching of the scar is to be prevented.

Skin stitches are removed in from three to five days for we prefer slight gaping of the wound to the unpleasant cross scar produced by the cutting through of a stitch.

The secondary procedures may be grouped under three main headings.

(1) General Contour Readjustment.

The commonest contour deformity seen in old hare-lip and cleft-palate cases is flatness of the lip and depression of the nose.

The flat lip is caused by lack of forward projection in the underlying bone and is most marked when the premaxilla has been removed.

The type of depressed nose most frequently encountered is one which is set back as a whole and one whose general line would, if prolonged, meet the vertical axis of the body at a more acute angle than that of the normal nose.

I need not refer to the factors responsible for this latter deformity, but we are of opinion that the scar tissue contraction in an antero-posterior direction which follows upon many cleft-palate closures plays a very important part in its production.

As a natural result of the backward displacement of the maxillæ, the upper teeth lie well inside those of the lower jaw, creating inefficient mastication and an over-prominent lower lip.

The operative procedure which we employ in such cases has been called "the epithelial inlay." It consists of freeing the lip and nose from the underlying retroposed bone, epithelializing the raw surfaces of the pocket thus produced, and later filling this skin-lined cavity by a projection from the upper denture sufficiently prominent to give normal contour and carrying artificial teeth, well in front of any normal teeth present, which occlude normally with the lowers.

The Thiersch graft used is applied on a mould held in position by a special dental splint or fixed to an already existing denture. It need scarcely be mentioned that the graft should be hairless.

When the nose position as a whole is not seriously affected, the undermining is not carried out behind the alar bases but is confined to the central part of the lip and the columella.

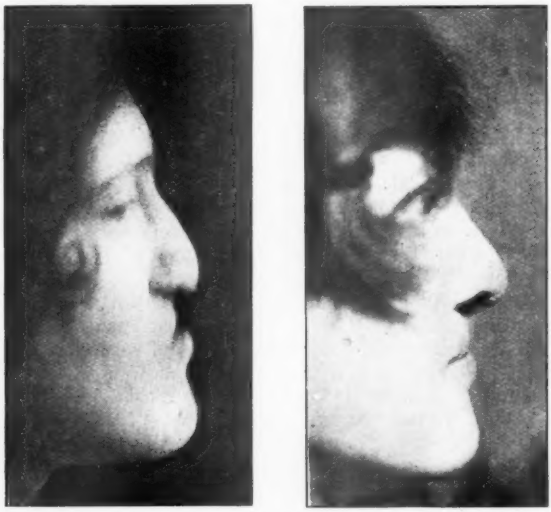


FIG. 1.—Old hare-lip case. Improvement in general facial contour obtained by "epithelial inlay" operation.

When the tip of the nose is dragged down and back, the incision in the mouth is carried forwards in the membranous septum and the lip and columella are thus made free to slide forwards on the antero-inferior border of the septum. The membranous septum is reconstructed by suturing the columella in its corrected and advanced position.

There are cases in which we carry the skin freeing still further, obtain access to the bony bridge and reduce any undue prominence or breadth by filing, or, fracturing the nasal bones after lateral saw and chisel cuts, reset the whole line of the nose.

Under this heading, however, the main point is the striking improvement in general contour obtained by "the epithelial inlay." (Fig. 1.)

(2) READJUSTMENT OF THE LIP.

I have already mentioned the difficulty we have experienced in determining the extent to which the prolabial skin should partake in the lip reconstruction. In most

cases coming up for secondary operation, there can be little question that this skin triangle has been incorporated too low down. There are still many surgeons who, led astray by its pseudo-vermilion margin, make it take part even in the lip margin itself.

In all cases where the nose tip is drawn down it is imperative to take the prolabial skin out of the lip and suture it so high upon the free border of the septum as will allow the tip of the nose to come forward into normal position. The "V"-shaped defect in the lip is closed by simple approximation, the deep tissues being built forward by deep catgut sutures to form a prominence at the base of the columella. This procedure draws in the alar bases and thus improves the nose still further.

We are quite prepared to admit that, as a secondary operation on a unilateral cleft, the usual excision of the scar line by incisions of the Rose type, which equalize

FIG. 2.—Result of straightening the nose and performing the "Cupid's bow" operation on the lip margin.

the length of the skin margins to be approximated, gives a passable result, but, aiming at a higher standard of cosmesis, we almost always proceed to what we have called the "Cupid's bow" operation.

A lip, broad from above down, straight and flat from angle to angle of the mouth, and with very little mucous membrane showing, is ugly and leaves little doubt as to the origin of the deformity.

The lip produced by the "Cupid's bow" operation is of very pleasing non-surgical type and loses once and for all its hare-lip characteristics. (Fig. 2.)

The existing skin-vermillion junction is discarded altogether and a new curved lip border incision is made at a higher level; the skin between this and the existing junction line is removed; and the mucous membrane, after slight undermining, is drawn upwards and sutured to the new skin edge. The result is an

attractive short lip, with full mucous membrane, and at least a suggestion of a "Cupid's bow." Incidentally, the lip margin is lengthened by the operation and becomes looser and in better proportion to the lower lip.

(3) Readjustment of the Nose.

In some cases nose reduction is called for; in others, the whole line of the nose is deviated to one side or the other, and nothing short of fracturing the nasal bones and resetting the whole bony bridge and septum will give a good-looking result.

Either of these procedures may be carried out, as has been indicated, in combination with one or other of the lip operations. It should be noted that resetting the bony bridge and correcting the deviation of the septum will often correct asymmetrical nostrils which resist all other forms of treatment.

The operation for depressed tip has been considered in dealing with the problem of the prolabial skin.

In some cases, apparently owing to retarded septal development, the nasal bridge remains seriously depressed. In these cases we usually carry out a cartilage graft from the ribs.

The various procedures for correcting the flattened or kinked nostril are well known.

In cases of serious asymmetry of the level of the nostrils, where nasal reposition has failed, we use a central columella incision, free the central crura of the alar cartilages well into the lobule of the nose, and, sliding them one on the other, suture the edges of the incision in corrected position and carry out any small local excision required for the correction of the slight secondary deformity which this shift produces in the tip of the nose.

Mr. C. W. Gordon Bryan.

I prefer to do the operation within the first three months of life, preferably at the age of three weeks. A point to which I attach the greatest importance is raising the soft parts on the inner side of the cleft very freely, right up round the columella and ala nasi, well into the cheek; in this way the nostril is closed by bringing the inner towards the outer side. I do not find that there is excess of tissue when closing the nostril if the soft parts are freely mobilized and the ala nasi of the outer side sufficiently rotated upwards, inwards and forwards, and I do not remove tissues therefore to make the nostril smaller. In closing the lip, in accordance with the practice of previous speakers, I cut away no tissue but use the red margin of the cleft as a support behind the suture line. At the primary operation I aim at a lip of good thickness and if subsequently it proves to be excessively long and flat make use of the "Cupid's bow" operation; this little operation I have found of the greatest value in producing a pleasant face from an otherwise ugly one.

Section of Urology.

President—Mr. Frank Kidd, M.Ch.

[*April* 26, 1928.]

Specimen of Papillo-Carcinoma of Ureter from a Male, aged 58.

By Jas. B. Macalpine, F.R.C.S.

In August, 1927, the patient began to suffer from pain in the suprapubic region and in the right loin. The pain in the back was intermittent and colicky.

In October he first noticed hæmaturia, following severe spasmodic pain in the bladder. From that time the hæmaturia was almost constant and at times was moderately profuse. There was no frequency of micturition.

The skiagram was negative.

Urine: In the absence of hæmaturia the urine contained no abnormal constituent.

The patient was referred to me in January, 1928, and on January 27 a cystoscopy was performed.

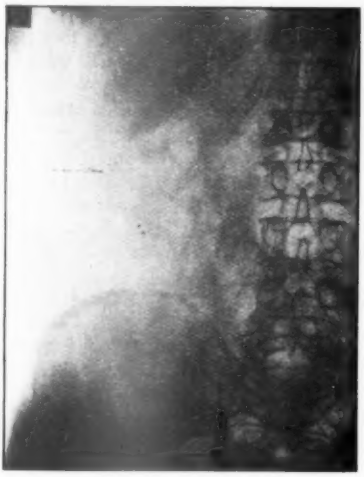


Fig. 1.

Fig. 2.

The bladder was normal in appearance, except that it was congested over the right side of the trigone and around the right ureteric orifice.

Indigo-carmine, injected intravenously, appeared at the left ureteric orifice in three and a half minutes with a good efflux; the right side was observed for seven minutes, during which no efflux was seen. A ureteric catheter was passed up the right side, but about four and a half or five inches up that tube it was obstructed.

A pyelogram (fig. 1) showed that no fluid passed beyond a point half an inch higher than the tip of the ureteric catheter (level of the sacro-iliac synchondrosis). A thin tongue was observed extending up from the ureteric tip, but the majority of the fluid returned immediately towards the bladder.

66 Macalpine: *Specimen of Papillo-Carcinoma of Ureter*

Operation, January 31, 1928.—The ureter was sought through an incision parallel to and just above Poupart's ligament. It was found to contain what appeared to be a growth and was dilated above that point. It was doubly ligatured and divided close to the bladder and the ureter was freed as far as possible towards the kidney. The kidney was subsequently removed through a lumbar incision.

Recovery was uneventful.

The pathological specimen (fig. 2) shows a papillo-carcinoma blocking the ureter completely, together with dilatation of the proximal ureter and of the renal pelvis.

The microscopical slides show a papillo-carcinoma.

REMARKS.

(1) The points suggestive of growth rather than stone as the cause of obstruction are: (*a*) The degree of the hæmaturia. (*b*) The completeness of the ureteric obstruction which is not characteristic of a stone.

(2) The ureter in such cases should be removed right down to the bladder, in order to ensure that no implants from the original growth are left behind. There was no evidence that the growth occurring at the sacro-iliac synchondrosis was the highest or the primary growth. It might itself have been an implantation.

Specimen: Fibrinous Body found in the Perivesical Tissue.

Shown by CYRIL A. R. NITCH, M.S.

THIS extraperitoneal perivesical tumour from the Museum of St. Thomas's Hospital is thus described in the catalogue:—

"A firm globular tumour, 2½ in. in diameter, which was removed after death from the subperitoneal tissue between the bladder and the rectum, loosely connected with the outer surface of the latter. It is of firm consistence and, except for a very fine concentric striation, would be homogeneous to the naked eye. In its centre, which is calcified, there lies a slightly curved eroded piece of steel or iron. Histologically the new formation consists of concentric lamellæ of bright, stiff, fibrous-looking tissue, for the most part devoid of recognizable cells. There are nowhere any traces of blood pigment."

At a recent meeting Mr. Clifford Morson showed a similar tumour which had been found at a post-mortem examination lying loose in the peritoneal cavity, and which was thought to be due to the deposit of fibrin on a detached appendix epiploica. In the present case the formation is identical, but the tumour has an obvious nucleus of metal.

Three Cases of Papilloma of the Ureter; Ureterectomy subsequent to Nephrectomy for Papilloma of the Renal Pelvis.

By Sir JOHN THOMSON-WALKER, F.R.C.S.

(I) DR. M., aged 65. One attack of hæmaturia six weeks before examination. No enlargement of kidneys. X-ray examination negative. Bladder normal on cystoscopy. Urine normal. The ureters were catheterized, and the urine from the left kidney contained 1·2 per cent. of urea; that from the right kidney contained

0·7 per cent. of urea. Suspicion therefore fell on the right kidney as the source of the hæmorrhage and pyelography was performed on this side.

The pyelogram showed a large papillomatous growth filling the pelvis with a narrow line of opaque fluid in the remaining lumen. Two dilated calices.

Right Nephrectomy (June 10, 1925).—Renal pelvis filled with large papillomatous growth. Ureter normal on palpation, cut and ligatured at level of lower pole of kidney. Pathological report: Simple papilloma of renal pelvis.

Two years and three months after operation (October 7, 1927) there was a severe attack of hæmaturia. Cystoscopy showed a large papillomatous mass occupying the right side of the trigone and covering the right ureteric orifice. Suprapubic cystotomy and removal of the papilloma. The right ureter was palpated and found distended with papilloma. Ureterectomy was performed through the median suprapubic incision.

Pathological Report.—Simple papilloma of the bladder and ureter. Convalescence rapid.

(II) Captain T., aged 65. Intermittent hæmaturia for seven months. Discomfort in left loin but no enlargement of the kidneys on palpation. An X-ray examination showed the left renal shadow slightly larger than right. No abnormality in urine from either kidney.

Pyelogram, Left Kidney (February 28, 1927).—17 c.c. sodium iodide. Filling defect shows silhouette of papilloma of upper wall of renal pelvis.

Left Nephrectomy (March 2, 1927).—Papilloma of the renal pelvis felt like a marble through the wall of the pelvis. Ureter normal on palpation of lumbar segment. Cut at lower pole of kidney and ligatured.

Four months after operation attack of hæmaturia. Two months later a second attack with pain in nephrectomy scar and left groin.

Bladder and ureteric orifices healthy. Catheter passed up left ureter produced no hæmorrhage. Pyelography of right kidney renal pelvis and calices normal. Ureterogram of left ureter filling poor, outline indefinite.

Left Ureterectomy (January 19, 1928).—Median incision. Upper end of ureter at level of third lumbar vertebra expanded and densely adherent. Pelvic segment of ureter normal.

Pathological Report.—Upper end of ureter expanded by papilloma undergoing malignant change and infiltrating underlying tissue of ureteral wall.

Rapid convalescence.

(III) Mrs. R., aged 68. Pain left renal region and severe hæmaturia for six months. Patient anæmic, breathless and giddy. Under treatment for myxœdema. Kidneys not enlarged on palpation.

Cystoscopy.—Bloody efflux from left ureteric orifice. Had been refused operation by a surgeon on account of heart and general condition.

Pyelography.—Left renal pelvis shows filling defect. Diagnosis, papilloma of renal pelvis.

Left Nephrectomy (January 12, 1926).—Small papilloma of renal pelvis.

Pathological Report.—Simple papilloma.

One year and nine months after the operation (October, 1927) attack of hæmaturia followed by intermittent attacks.

Cystoscopy showed pure, dark blood oozing out of left ureteric orifice at intervals.

Left ureterogram showed dilated upper end of blind ureter with appearance of a filling defect at the level of the upper border of the fourth lumbar vertebral body, and another in the course of the ureter at the level of the fifth lumbar vertebra.

Left Ureterectomy (March 13, 1928) by median incision. Upper segment of ureter expanded with papillomatous growth; lower end normal. Uninterrupted recovery.

68 Thomson-Walker: *Three Cases of Papilloma of the Ureter*

Discussion.

These three cases are consecutive cases of ureterectomy, and they point to the frequency with which the ureter becomes the seat of implantation in cases of papilloma of the renal pelvis.

It is of interest to note that at the time of the operation the lumbar segment of the ureter appeared to be quite normal so far as could be ascertained by inspection of the cut end and palpation of the duct. Yet this was the part of the ureter that was most seriously affected in two of these cases.

There are several points of interest in regard to diagnosis of papilloma of the ureter in these cases after the nephrectomy. Pain was present in one case, and on one occasion was situated in the nephrectomy scar and in the groin.

In one case blood was seen oozing in a treacly stream from the ureter on the nephrectomized side.

The type of hæmaturia was peculiar. The blood passed was not mixed with the urine, but was passed either before the urine or after it, and the urine passed in the rest of the micturition was quite clear. The blood settled in a separate layer in the specimen glass.

In one case a papilloma of the bladder covered the ureteric orifice of the nephrectomized side, and the inference was justifiable that papillomata were present in the ureter also. This was confirmed when the bladder was opened and the ureter palpated, the lower ureter being thickened and filled with growth.

In one case no bleeding was present on two examinations, and there was no indication as to whether the bleeding came from the ureter of the nephrectomized side or from the remaining kidney or its ureter. A catheter passed vigorously up and down the lumen of the ureter stump was not followed by an issue of blood from the ureteric orifice. In this case it was possible that a papilloma might be present in the pelvis of the remaining kidney. Pyelography of the remaining kidney was therefore carried out and was negative.

This raises the important question as to the value of a normal pyelogram in the diagnosis of growth of the kidney.

I have watched this point in diagnosis for some time, and have not met with a case in which a pyelogram showed a normal pelvis and calices when papilloma of the renal pelvis was present. But there may be difficulties when the pyelogram, as a result of imperfect bowel preparation or the presence of extensive calcification of rib cartilages, or from the poor quality of the radiogram, gives an indefinite or an apparently negative result.

In one case very definite filling defects were seen in the ureterogram of the ureter stump, and gave clear evidence of the presence of growth in the ureter. So far as I know, this has not previously been demonstrated.

An obvious criticism of these cases is that the ureter should have been removed at the time of the nephrectomy.

This point was considered, but all these patients were in a very poor condition and there was nothing at the time of the nephrectomy to show an extension down the ureter.

One of the patients had been refused nephrectomy by another surgeon on account of her circulation and general feebleness. Another patient was regarded as a very poor subject, and his physician was originally opposed to removal of the ureter at the same time as the bladder papilloma, and agreed only during the operation.

So that in these cases a complete ureterectomy at the time of the nephrectomy was not considered justifiable.

There must, however, be cases in which the patient's condition is good, and in these nephro-ureterectomy is certainly indicated.

Cases, Specimen, &c., were also shown by:—

Mr. FRANK HARVEY: Drawing of Ligature found in the Bladder after Hysterectomy.

Mr. J. B. MACALPINE: Cystin Stones from Identical Twins.

Mr. SYDNEY MACDONALD: (1) A Pulmonary Embolus (following Nephrolithotomy). (2) Alleged Urinary Calculi.

Mr. E. T. C. MILLIGAN: (1) Case of Pseudo-Elephantiasis of Penis and Scrotum: for Diagnosis. (2) Tuberculosis of Horse-shoe Kidney.

Mr. ALEX. E. ROCHE: Case of Orchidopexy into Scrotum of Femoral Testicle.

Mr. KENNETH WALKER: (1) Torsion of the Testicle. (2) Large Ureteric Calculus.

Mr. H. P. WINSBURY WHITE: (1) Uretero-nephrectomy for Stones in the Ureter. (2) Foreign Body from Female Bladder.

[*January* 26, 1928.]

Teratoma of Kidney.[1]

By SYDNEY MACDONALD, F.R.C.S.

F. P., MALE, aged 50. From the age of 11 until that of 24, the patient suffered from attacks of pain in the left loin, working round to the front and groin, and during these attacks passed "discoloured water." From the age of 24, up to August, 1927, he was quite free from pain, but from this date up to the time of operation in December, had frequent attacks, chiefly at night. Beyond the passage of a few small clots on one occasion there was no further hæmaturia.

On examination the left kidney was enlarged, the urine clear and free from pus; an X-ray examination revealed a stone, the size of a large cherry, in the kidney pelvis. Catheterization of the right ureter showed a satisfactory kidney on this side, and nephrectomy was accordingly carried out on the left. The patient made an uneventful recovery.

Specimen [reported by Dr. Elworthy].—A hard, solid, irregularly rounded tumour, weighing 650 grm. and measuring 12 by 10 by 11 cm., occupies the upper two-thirds of a hydronephrotic kidney whose pelvis contained a stone. Its outer and pelvic aspects are coarsely bossed and a lobulated extension offsets into the hilic fat parallel to the ureter. The cut surface fails to show any normal kidney. A tough, somewhat translucent, white tissue supports areas that are cream-coloured, opaque and friable. Localized hæmorrhages occur.

Microscopically, the bulk of the tissue consists of interlacing bundles of leiomyomatous cells, which in parts are very similar in histological appearance to a myoma of the uterus. But in other places, roughly corresponding to the cream-coloured friable areas of the macroscopic description, the cells are more loosely and less regularly disposed, and are more polymorphous and neoplastic in character. The myomatous tissue is roughly partitioned into lobular masses by septa sinking in from the fibro-cellular capsule.

Embryonal tubules occur in these septa, and also amongst the myomatous cells. They are lined with short cubical epithelium, are supported by a meagre scaffolding of fibrous tissue and usually show a well-defined lumen. Their number at any one

[1] The publication of this report has been held over in order to include illustrations.

point is never large, the myo-sarcomatous tissue providing the preponderant bulk of the tumour.

The following points were also noted:—

Remnants of the ruined kidney were distributed around the periphery of the tumour.

A few adult blood-vessels were found, but for the most part simple channels lying amidst the myomatous cells were the rule. There appeared to be no particular relationship between the architecture of the growth and blood-channels. Areas of moderate leucocytic infiltration were present in many parts, as were also interstitial hæmorrhages and patches of necrosis.

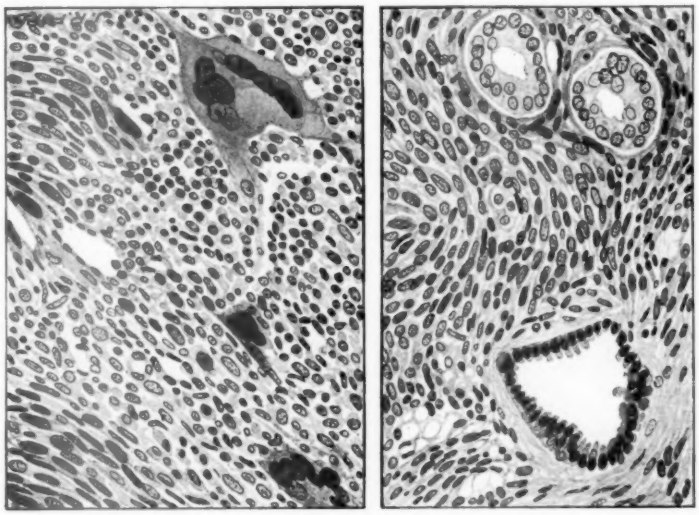
FIG. 1.

FIG. 2.

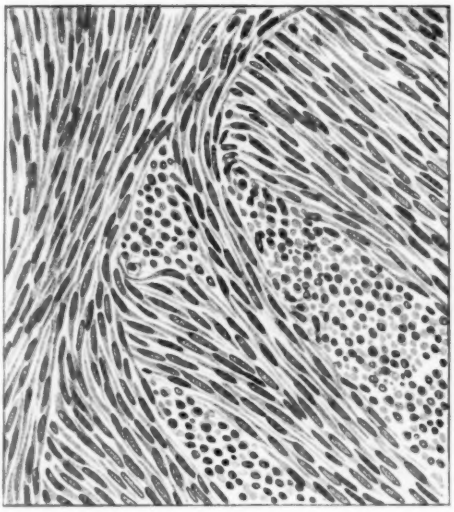
In one place cells indistinguishable from round-cell sarcoma were found. Giant cells were present in small number. Cross striation could not be demonstrated in any of the cells.

It was concluded that this specimen was a mixed tissue tumour of the kidney, malignant in character, and closely related to the embryonal adeno-myo-sarcomata of infancy.

POSTSCRIPT.—The patient died four months after operation. A large recurrence occupied the bed of the left kidney. This had spread upwards behind the peritoneum to form large, soft white masses burying the pancreas and pushing forward the liver. Above the diaphragm the posterior mediastinum and right thorax were filled with growth to about one-third of their extent. True metastases were rare. One of large



(*A*) (*B*)



(*C*)

FIG. 3. Drawings from microscopic sections.—(*A*) a giant-cell-bearing area; (*B*) embryonal tubules; (*C*) interlacing bundles of leiomyosarcomatous cells.

size was noted in the middle of the left lobe of the liver, and another—insignificant in dimension—in the manubrium. A few nodules of growth occurred within the peritoneum attached to omentum and coils of intestine in the immediate neighbourhood of the local recurrence in the left renal pouch.

The right kidney, the inferior vena cava, and the pelvic viscera were free.

The histology of the recurrent growth was as shown in figure 3, A and C, definite tubules not being apparent.

Mr. FRANK KIDD (President) said that teratoma of the kidney was a very interesting condition. He could not recall having seen one, except in a child. This patient had probably had it in the kidney since childhood, growing very slowly, and when a stone had destroyed the kidney, the teratoma had been discovered accidentally.

Section of Laryngology and Section of Otology.

COMBINED SUMMER MEETING, MAY 31, JUNE 1 AND 2, 1928.
OTOLOGICAL SESSION, JUNE 1, 1928.

Chairman—Mr. J. S. FRASER, F.R.C.S.Ed.

The Pathology of Otosclerosis.

By OTTO MAYER, M.D. (Vienna).

IT is well known that the deafness in cases of otosclerosis is chiefly due to ankylosis of the stapes, caused by disease of the bone of the labyrinth capsule. Such disease appears in sharply outlined areas (foci) localized to definite situations and most frequently occurring in the neighbourhood of the oval window. They are, however, often found in the neighbourhood of the attachment of the round window membrane and, less frequently, in the region of the internal auditory meatus and the semicircular canals.

How do these diseased foci develop? The original bone of the labyrinth capsule becomes absorbed by osteoclasts and a bone-marrow is formed, in which an excessive number of osteoblasts abound. In this marrow the new bone is formed, but is imperfect. It is reticulated, stains dark blue with hæmalum and eosin, and contains a great number of large and irregularly situated bone-cells. This imperfect new bone becomes gradually surrounded by more perfect bone. Such tissue as the blue-staining bone is never seen in the normal labyrinth capsule or in other pathological conditions. It is this fact which gives us the key to the character of this particular disease, since it can thus represent neither a physiological transformation nor an inflammatory development. Physiological transformation of bone develops in quite a different way—never in discrete foci but always in irregularly dispersed canal systems. In physiological transformation and in inflammatory conditions, new blood-vessels enter the canals of the old bone.

Further, in otosclerosis there is no exudation such as occurs in cases of inflammation, and the bone spaces contain not granulation tissue, but an osteoblastic marrow. Manasse, it is true, considered that this osteoblastic marrow might be granulation tissue, but this cannot be the case since it contains no leucocytes or plasma cells.

In my opinion otosclerosis should really be regarded as a hyperplasia. This view is supported by the following facts: (1) The new-formed bony tissue in an imperfect atypical tissue, never found in the labyrinth capsule in other conditions and always of a distinct elementary type. (2) The new bone shows marked

histological variations; for instance, it may be extremely sclerotic and very poor in marrow spaces, or, on the other hand, marrow spaces filled with osteoblasts may be its chief feature. Further, a fibrous form is found in which large portions of the bone are filled with fibrous marrow containing very few cells. In still other cases cystic spaces occur as the result of absorption of the marrow. Lastly, there may be a vascular formation in which the marrow spaces are almost completely occupied with large vessels. Such histological variations are of course a feature of hyperplasias and tumours.

The Method in which these Diseased Areas Grow.

(*a*) They may appear on the upper surface of the labyrinth capsule, on the wall of the tympanic cavity, or on the walls of the various spaces of the labyrinth. Thus the oval window and the niche of the round window may become completely occluded. Internally, exostoses are most frequently found in the peri-lymphatic space of the vestibule in cases in which the growth on the upper or lower border of the oval window displaces the stapes inwards. Exostoses may also appear in the scala tympani of the basal coil of the cochlea, filling it up almost completely; such an instance is especially noticeable in one of my own cases, in which the growth spreads upwards into the cochlea, actually pressing the bony partition between the lateral wall and the wall of the apex into folds. We seldom see such exostoses on the surface; here the labyrinth capsule is merely invaded by the new bone, whilst maintaining its original contour.

(*b*) We must regard the growth of these diseased foci as a tumour, since it begins as a swelling of the marrow, followed by ossification.

(*c*) The overgrowth also involves the adjacent fibrous tissue, which at first becomes ossified and then undergoes a transformation. Thus we see the annular ligament of the stapes and also the ligamentum spirale becoming involved in this overgrowth.

(*d*) A most important fact in determining the character of these diseased areas in otosclerosis is their multiple appearances, for we know that this is also a characteristic feature of hereditary lesions and some other hyperplasias. I would remind you especially, in this connexion, of the multiple exostoses which appear spontaneously as a hereditary lesion scattered throughout the whole skeletal system and traceable to maldevelopments of that system. I would also instance the condition of multiple neuro-fibromata which likewise may occur over the whole peripheral nervous system or in special parts of it, and must be regarded as a localized manifestation of some general systemic disease.

(*e*) Similarly, there is also a definite symmetry of these diseased areas, which roughly occur in identical positions on both sides. It may of course be urged that inflammatory lesions may also leave symmetrical scars, for instance in the tympanic membrane. This is indeed the case, but such symmetrical inflammatory lesions are the exception, whilst in otosclerosis symmetry is the rule. A similar symmetry is also found in connexion with maldevelopments and tumours.

(*f*) A further strong support of this theory is the typical localization of the areas of otosclerosis. As I have previously remarked, a focus of otosclerosis is constantly found in that part of the labyrinth lying in front of the oval window. For a long time we did not understand why this was found in every case. Through my investigations this fact has been explained, for I have shown that in this situation there exists a synchondrosis in the external periosteal aspect of which this area of otosclerosis commences. This is important because, of course, exostoses develop in

similar situations in other parts of the body. I have been able to demonstrate small areas of atypical bone in synchondroses in many cases. These areas of atypical bone I regard as a deformity of the tissue, from the growth of which the otosclerotic foci really arise. Such atypical bony tissue is also found elsewhere in connexion with the areas of otosclerosis, more especially on the lateral aspect of the attachment of the round window membrane. Further, the areas of atypical bone distributed throughout the region of the semicircular canals are really only similar deformities of the tissue. The occurrence of such deformities of tissue in the labyrinth capsule is intelligible, since the capsule remains during life in an embryonic or arrested state of development (Lange). In this respect the labyrinth differs from all the other bones of the body laid down in cartilage.

I maintain that these data, confirmed by my investigations, authorize my view that the otosclerotic areas in the labyrinth capsule are really pathological growths which arise in connexion with embryonic maldevelopments. One could indeed include them with the hyperplasias which are the result of faulty development or lack of development. Thus, according to this hypothesis, the areas of otosclerosis result from a growth of malformed embryonic osteoblastic tissue which usually occurs in definite positions of the labyrinth capsule. In these areas the pre-existing bone becomes absorbed and new bone is formed.

Besides the anatomical distribution of these areas, there are many other facts which support my theory. Firstly I would remind you that we find in otosclerosis other hyperplastic bony growths in the petrous bone, e.g., exostoses on its posterior aspect in the internal auditory meatus or in the tympanic cavity.

According to Hammerschlag, a noticeable point in the consideration of otosclerosis is the occurrence of areas of the disease in connexion with disturbances of development of the inner ear. (Alexander has found hyperplasias of the inner ear in cretins.) In fifteen cases of congenital deaf-mutism or difficulty in hearing dependent on congenital maldevelopment of the inner ear examined by me, I found three instances, i.e., 20 per cent., with typical areas of otosclerosis. Such a high percentage excludes accidental coincidence since areas of otosclerosis are only to be found in 5 per cent. of all temporal bones examined. Further, I found in two other cases a disease similar to otosclerosis and in one case multiple exostoses in the external auditory canal, whilst in only nine cases of those fifteen could no disease of the bone be discovered. You will thus see that in 40 per cent. of the cases the bone showed pathological changes which could be regarded as deformities of the tissue and that in 20 per cent. of the cases there were definite areas of otosclerosis. On the other hand, I found in fifteen out of the thirty temporal bones affected with otosclerosis which I examined slight maldevelopment of the inner ear and anomalies in the structure of the cochlear framework, more especially of the modiolus. Fischer and Brunner have each found deformities in otosclerosis. Such anomalies, according to Siebenmann and Nager, are characteristic of the hereditary and family form of deaf-mutism and deafness. Alexander and Tandler found similar anomalies of the modiolus in dancing-mice.

The establishment of this fact is especially interesting because we know, from the work of Bezold, Hartmann, Hammerschlag and Körner, that otosclerosis is a definite hereditary disease. Thus this circumstance, already recognized clinically, is supported by the conditions found in the inner ear by pathological investigation.

In this connexion I would remind you that hyperplasias such as multiple exostoses are hereditary. These multiple exostoses distributed throughout the skeleton are regarded as degenerative manifestations and are found in individuals who also show other degenerative stigmata. It is noteworthy that patients suffering

from otosclerosis show degenerative stigmata with great regularity (Bauer and Stein).

As regards the atrophy of the labyrinth almost always found in connexion with otosclerosis, I hold that the areas of otosclerosis themselves are only in a few cases the cause of the affection of the inner ear; in most cases no such connexion can be demonstrated. More often the cause consists in the fact that in the inner ear of patients suffering from otosclerosis there is a lack of development which is demonstrated firstly by certain malformation and secondly by a peculiar tendency of the connective tissue of the inner ear to become ossified. This peculiarity leads to a faulty arrangement of tissue and terminates in a gradual atrophy of the labyrinth.

All these points force us to conclude that otosclerosis affects not only the labyrinth capsule, but also the whole auditory apparatus.

We must imagine that in cases of otosclerosis the whole auditory organ has a morbid tendency and that the labyrinth capsule is especially affected. The latter, during life, consists, for the most part, of embryonic tissue and therefore a disposition to malformations persists to the latest age. Such maldevelopments, localized as they are to particular places, become out-growths of bone and often take the form of exostoses or hyperostoses. The labyrinth itself has similarly a morbid tendency, as is evidenced by the maldevelopment of the framework of the cochlea, and the tendency to ossification of its connective tissue. It is this morbid tendency which is the origin of the atrophy of the labyrinth.

Thus the disease of the labyrinth capsule, as well as that of the inner ear, have a common origin in an hereditary tendency.

Why in one case embryonic maldevelopments should be represented by an overgrowth, whilst in other cases no such tendency occurs, is as little known as is the cause of the development of tumours generally, and is at present completely inexplicable. It is probable that the matter can be referred to the influence of the endocrine secretions. Clinical experience seems to show that the areas of otosclerosis begin to increase during pregnancy, but we do not know anything definitely about this point. It is, however, worthy of note that during pregnancy overgrowth may be observed in other bones—the so-called puerperal osteophytes. It is also known that osteomalacia occurs during pregnancy, and this circumstance has led us to consider the possible connexion of otosclerosis with disturbance of the internal secretions. (Denker, Frey, Voss, Leicher.) Examinations, however, carried out in this direction have not yet afforded a definite result. On the other hand, it is quite certain that otosclerosis shows no relationship with osteomalacia or rickets, as certain American authors have believed (Bryan).

Another disease of bone, however—the osteitis deformans of Paget—has a connexion with otosclerosis. Even during my earliest investigations of otosclerosis I was struck with the similarity of this condition to that found in osteitis deformans. Later, I examined several temporal bones from patients who had suffered from Paget's disease and found that transformation of the bone of the labyrinth capsule occurred in a similar way to that found in otosclerosis, i.e., the original bone becomes replaced by a new formation, but the contour of the labyrinth capsule for the most part remains undisturbed. Sometimes, however, exostoses are formed and invade the labyrinth, or the labyrinth may be partly filled by new formations of bone-marrow. Lastly, an ankylosis of the stapes may develop.

The difference, however, consists in the fact that in Paget's disease the lesion is not restricted to localized areas, but is diffuse, and further, that the new formed bone is better developed; still, I have been impressed by the fact that in some instances of Paget's disease there were localized lesions similar to those found in otosclerosis.

Otosclerosis is, of course, very common, and thus one cannot absolutely exclude the factor of accidental coincidence, but it seems to me very probable that there is some connexion between the two diseases. Unfortunately we know little with certainty of Paget's disease, except that, according to the latest investigations, the hypothesis of Recklinghausen, as to its being an inflammatory lesion, can no longer be regarded as correct. Reinhardt considers that it is a disease of the connective tissue of the bone, and instead of the term osteitis fibrosa would propose to call it fibromatosis osteoplastica ossium.

A most important fact is the combination of otosclerosis with blue sclerotics and osteopsathyrosis. We owe the knowledge of this complication especially to Dighton, Fraser, Bronson and De Kleyn. This combination is explained by the fact that otosclerosis, as well as blue sclerotics and fragilitas ossium, can be traced back to a constitutional anomaly of the connective tissues (Bauer). This was especially noticeable in the following case, which I observed and histologically examined.

The patient in question was a woman of 54 years, who suffered from otosclerosis and blue sclerotics and was operated on for carcinoma ventriculi. During the operation the surgeon noted an abnormal lacerability of the veins and also of the muscular coat of the stomach and intestines. The patient died of pneumonia, and at the autopsy we found a narrowness of the ureters and of the blood-vessels, as well as diastases of the recti muscles. Histologically, I found in both temporal bones an area of otosclerosis near the oval window and slight malformation of the inner ear.

These findings may be regarded as proving that, in addition to fragility of the bones, there are also other abnormalities of the mesenchyma. Otosclerosis, as Bauer said, may be placed in a group which is based on anomalies of the connective tissue. All these conditions, such as blue sclerotics, osteopsathyrosis, ostitis deformans, hypoplasia of vessels and otosclerosis are caused by changes in the connective tissue.

With this view the coincidence of areas of otosclerosis with tumours of the eighth nerve would be intelligible, as these tumours are neurofibromata. Thus, we are not surprised to find that the eighth nerve is the only one of the cranial nerves in which tumours occur, and that the labyrinth capsule is the only portion of the cranial bones subject to such multiple lesions. These lesions of the nerve and the bone are the result of similar maldevelopments of the connective tissue.

The result of all these investigations leads us to the conclusion that areas of otosclerosis are to be regarded as hyperplasias. This view is based not only on the histological appearance of the foci, but also on their multiplicity, their typical and symmetrical localization, the presence of minute islands of atypical tissue (constituting the points of origin in these areas), the simultaneous presence of maldevelopments in the inner ear and in other parts of the auditory organ, the condition of hyperplasia of the temporal bone generally, the appearance clinically of congenital features, the coincidence with blue sclerotics and osteopsathyrosis, Paget's disease, and neurofibroma of the 8th nerve, and lastly, the hereditary factor found in this disease.

Only on the basis of this view are we able to understand the whole clinical and anatomical picture.

With no other hypothesis is it possible to bring otosclerosis into the realms of general pathology, and whilst all other efforts to explain the condition postulate an entirely new variety of disease, I claim that my hypothesis agrees well with the recognized process of disease in other parts of the system.

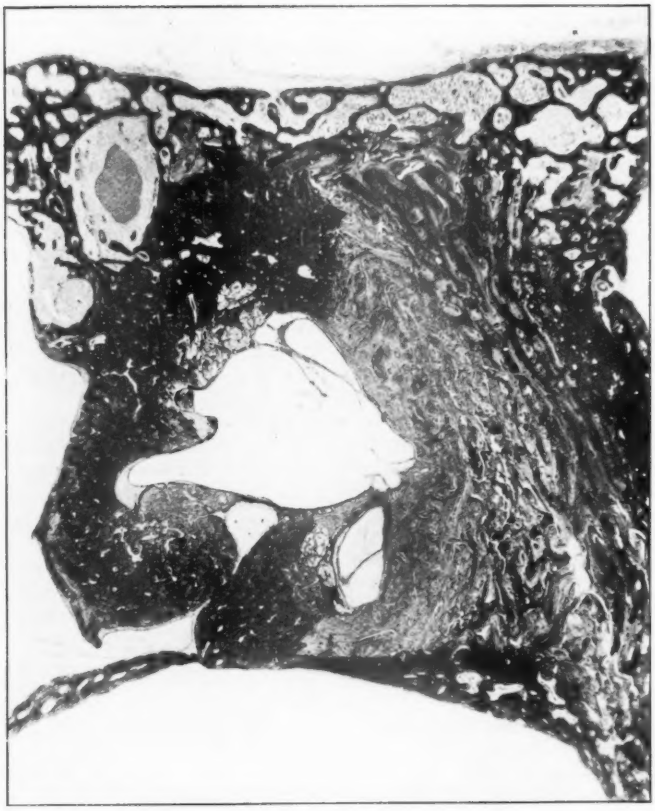


FIG. 1.

FIG. 1 shows a large area of otosclerosis in the region of the oval window. The area of otosclerosis has spread above the vestibule and has involved the whole promontory and greatly narrowed—or even obliterated—the niche of the round window. A further area of otosclerosis appears between the ampulla of the posterior canal and the round window, and, lastly, there is an area in the roof of the jugular bulb.

FIG. 2.

FIG. 2 shows otosclerosis in the upper and lower margins of the oval window, with ankylosis of the stapes to the upper margin. The stapes itself is affected and, further, there is an area of otosclerosis in the niche of the round window.

FIG. 3.

FIG. 3 shows extensive spongification of the petrous bone. There is an area of otosclerosis surrounding the facial nerve above the cochlea. There is a further area around the internal acoustic meatus.

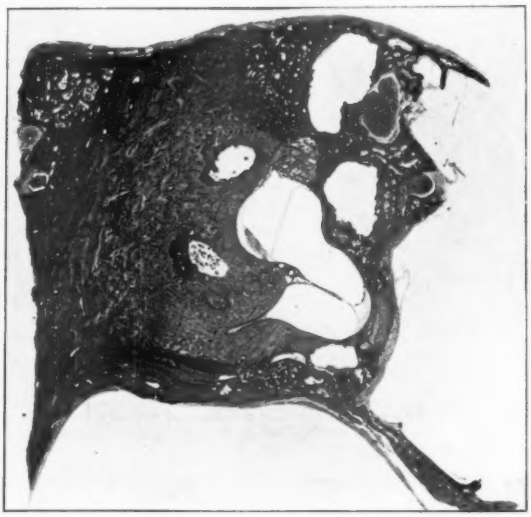


FIG. 4.

FIG. 4 shows areas of otosclerosis in the inner wall of the attic in the promontory, close to the branch of the vestibular nerve to the posterior semicircular canal. There are further areas in the postero-superior angle of the petrous and just above the jugular bulb.

FIG. 5.

FIG. 5 shows an area of otosclerosis in the anterior margin of the oval window, with a narrow area of ankylosis between the otosclerotic area and the anterior part of the footplate of the stapes. The stapes itself appears otherwise free from change.

FIG. 6.

FIG. 6 shows the bony capsule of the cochlea completely replaced by otosclerotic tissue which has invaded the scala tympani of the basal coil and obliterated it. The scala vestibuli of the upper part of the middle coil is also narrowed by the otosclerotic or spongified bone.

A Working Hypothesis for Research in Otosclerosis.

By A. LOWNDES YATES, M.D.

WHEN over a year ago I began to investigate the ætiology of chronic progressive deafness, I formed the opinion that it would be necessary to follow two lines of work, namely, (1) the establishment of a system of quantitative testing of the hearing which could preferably be expressed in graphic form, and (2) the estimation, in this way, of the various forms of deafness.

I have, as a result of this incomplete investigation, formed certain conclusions, and since the data on which they are based can be proved or disproved by any otologist, I thought it might be of interest to bring them to your notice.

The work of Professor Mayer in Vienna and that of Dr. Fraser (our President), Dr. Albert Gray, Mr. G. J. Jenkins, and Mr. Sydney Scott in this country has proved the nature of otosclerosis and the character of the alteration of the hearing when the essential lesions of the disease are present. Whether the lesions in the otitic capsule are the result of inflammation in the middle ear, of circulating toxins, of congenital peculiarities in the bones or of endocrine disorders, or are of an atrophic nature, is not only not agreed but there is no evidence in the literature to point the way to a conclusion.

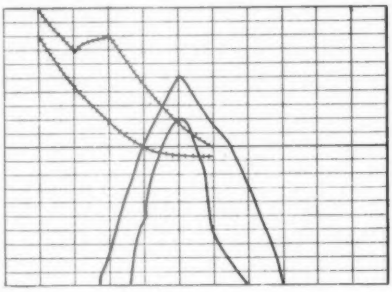


FIG. 1.

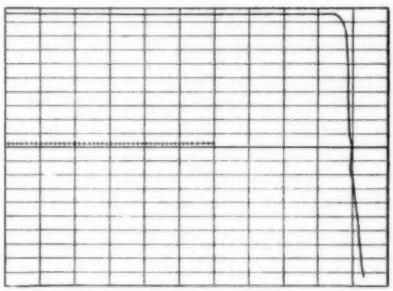


FIG. 2.

The work of Hartman goes to prove that if the disease affects the cochlear nerve, there is a progressive increase in the power of hearing of the lower tones as we pass down the auditory scale, and that, if the stapedio-vestibular junction is affected, the reverse is the case. If the lesion affects both situations he holds that there is a combination of both types of alteration of the hearing and that this is commonly the case.

I regard as cases of clinical otosclerosis those in which Bezold's triad symptom complex is present together with a patent Eustachian tube, and with no evidence of any demonstrable adhesive process in the middle ear or of a former perforation of the membrane. I found that the graph in these cases of clinical otosclerosis showed definite characteristics which are shown in fig. 1.

Fig. 2 is the graph of normal hearing.[1]

[1] For the graphs of the adhesive type of catarrhal otitis media, of nerve deafness, etc., and for the methods of preparation of these graphs the reader is referred to the author's paper on the subject, *Proc. Roy. Soc. Med.*, 1928, xxi (Sect. Otol.) 34.

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It has been the object of this investigation to discover the conditions which, when present in the ear, give rise to alterations in the hearing similar to those met with in the condition known as clinical otosclerosis.

Fig. 3 shows the graph of a case in which the onset was insidious, the first noticeable evidence of deafness having appeared five years ago. The patient became much worse some seven months ago. Two months ago she had an attack of very mild acute otitis media; temperature 99° F., with slight fullness and bulging of both membranes. Myringotomy was performed and was followed by a discharge which was mucopurulent. The myringotomy wound healed rapidly. A month afterwards there was an attack of right aural pain, but I could find little abnormality in the membrane; pus could, however, be sucked from the Eustachian tube with the Weber Liel catheter. She had similar attacks in the left ear also. About ten days later the patient had an attack of pain in both ears, and again pus was sucked from the Eustachian tubes. The interesting point is that the graph is substantially that of otosclerosis, but the case is one of subacute otitis media.

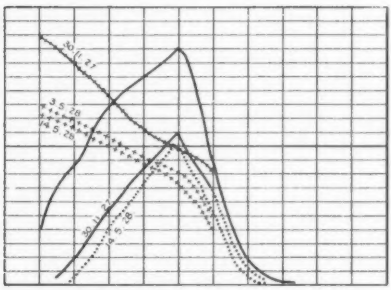

FIG. 3.

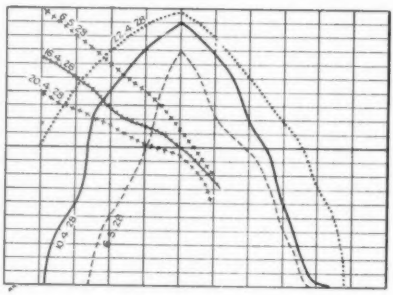

FIG. 4.

Fig. 4 shows the graph of a patient in whom tinnitus and aural pain first made their appearance during an acute febrile attack eighteen months ago for which quinine in moderate doses was administered.

She has had some tinnitus ever since and intermittent attacks of aural pain have been present periodically. When examined during an attack of aural pain the membrane was not altered in position. Pus was aspirated from the Eustachian tube and from the maxillary antra. The latter fact led to a complete exploration of the sinuses under anæsthesia, the antra being drained.

It was noticeable that during the attacks the graph took on the characters met with in otosclerosis and that each attack left her hearing a little more impaired than previously.

The intranasal drainage of the antra improved the hearing considerably, but tinnitus remained.

Fig. 5 shows the graph of a boy who, when at school, became deaf and had slight aural pain. Adenoids were present in the fossa of Rosenmüller, and at the time of their removal pus was sucked out from the Eustachian tubes, and the hearing later lost the characters of clinical otosclerosis and returned to normal.

Fig. 6 shows the graph of a boy who had an acute otitis media following an explosion in a laboratory. Pus was flowing from the perforation in the left membrane, but this did not at first adequately drain the tympanum. During the period of inadequate drainage the graph showed the characters of an early clinical otosclerosis, and later, when the drainage became adequate, these characters were lost and the hearing returned to normal.

I have records of many other cases in which the same similarity of the graph in subacute otitis media, acute otitis media, and clinical otosclerosis are exemplified, but the above will be sufficient to draw attention to the matter.

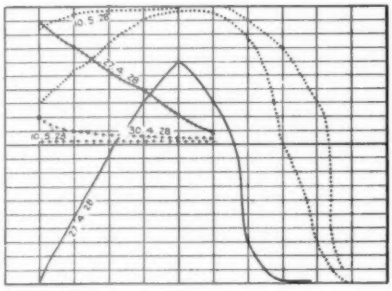

FIG. 5.

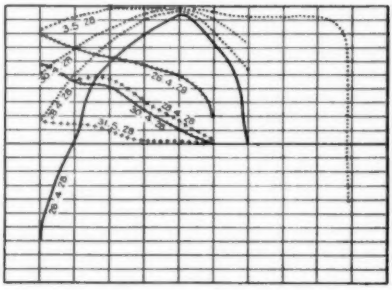

FIG. 6.

CONCLUSIONS.

From the point of view of audiographs there are three types of deafness:—(1) Nerve or internal ear deafness. (2) Otitis media with an indrawn membrane and adhesions. (3) Acute otitis media, subacute otitis media, and otosclerosis, in which a progressively increasing departure of the graph from the normal can be demonstrated.

This would seem to formulate the hypothesis that clinical otosclerosis is at times the terminal stage of subacute otitis media in which the products of inflammation of the middle ear are conveyed away by the Eustachian tube. If the case is more acute, and if a perforation forms in the membrane, the case is not considered clinically as one of otosclerosis, though the impairment of the hearing may be similar.

It is easy to formulate a theory which affords a reasonable explanation of the manner in which a chronic inflammatory process of the middle ear might bring about a pathological condition such as is met with in otosclerosis, but it is desirable to wait until the intermediate stages have been demonstrated pathologically.

Discussion.—Dr. ALBERT A. GRAY said it was interesting that Professor Mayer took the view that absorption of bone in otosclerosis took place by means of osteoclasts, since Manasse held the opinion that osteoclasts took no part in the absorption; he thought that the absorption of bone was by simple pressure. He (the speaker) agreed with Professor Mayer that osteoclasts took a part in the formation; some of his own specimens showed osteoclasts absorbing the bone.

He agreed with almost everything Professor Mayer had said. For instance, he himself also thought that otosclerosis was not an inflammatory condition. It was, in his view, a

degenerative process in the organ of hearing as a whole, and the tendency towards that process must be present in the individual for this condition to result. He did not think the condition was hereditary in every case.

Another important point which this demonstration brought out was that the change in the bone was not constant. Many thought there was absorption of a certain amount of bone and then a deposition of excess bone in its place, but that was not always so. In some cases no new bone was deposited, there was only absorption of bone. In others, the bone deposited was less than that absorbed. That was where the difficulty arose in attempting to associate the condition with osteitis deformans. In the latter disease an excessive deposition always followed absorption.

He also agreed with Professor Mayer that otosclerosis was a disease affecting the whole auditory apparatus, as he (the speaker) stated in his book in 1917. Diminished secretion of wax in the external auditory meatus and the striking diminution in the sensitiveness of the tympanic membrane and the skin lining the external auditory meatus could not be accounted for by changes in a minute area of bone in the capsule of the labyrinth. He went further than Professor Mayer and maintained that even the auditory nerve connexions into the cortex might be affected. Future research work should include not only the temporal bone, but also the auditory nerve tracts and the brain cortex. He hoped the International Committee which was working on this subject would ask not only for the temporal bones, but also for the brains of those who during life had suffered from this condition.

Otosclerosis was confessedly a mystery, and, like all mysteries, it possessed a great fascination.

Mr. G. J. JENKINS pointed out that osteitis deformans was, in some cases, identical with middle ear deafness, whereas in others it was of the internal ear deafness type. He had microscopically examined four specimens of osteitis deformans, and in none did he find any of the bone changes characteristic of otosclerosis. He did not think those two bone diseases were identical. He thought osteitis deformans caused the clinical aspects of "otosclerosis" by involving the same structures in a similar manner as did the other bone disease of otosclerosis.

Mr. SOMERVILLE HASTINGS said the two papers clearly illustrated the importance of having definitions before considering pathological conditions. Dr. Lowndes Yates defined otosclerosis as middle ear deafness with von Bezold's triad of symptoms; Professor Mayer defined it as a pathological condition showing changes in the capsule of the internal ear. That this definition could not correspond with Dr. Lowndes Yates' was shown by his statement that in many cases otosclerosis was associated with deaf-mutism, which could not possibly show von Bèzold's triad.

From the clinical side he was interested in Dr. Lowndes Yates' paper, as he believed he had himself seen such cases, which he would describe as typical middle ear deafness changing to what must be termed otosclerosis.

There was a tendency to conclude that because in otosclerosis no active changes in the middle ear, and no septic focus were to be found, there had not been any such changes; but an inflammatory process could be caused by trouble in one situation, and this trouble might disappear, but the process might persist. As Professor Mayer had shown, in these cases there was what closely resembled a tumour formation, but it was not justifiable to say that this could not be due to inflammatory changes. Enormous hypertrophies resembling tumour formations occurred in the lower jaw and dental surgeons said these were orginated by pathological changes due to unhealthy teeth. He (the speaker) did not see why a similar process should not take place in the capsule of the internal ear.

Mr. JAMES ADAM said he knew of three brothers, the youngest aged 46, the eldest 54. All had blue sclera, all had pronounced arcus senilis, and were grey-haired, but none had obvious atheroma of the arteries. In the two younger there was definite otosclerosis, and both had had suppurative disease in the middle ear. The eldest had not had middle ear disease and his hearing was normal. He had two daughters, neither of whom had suffered from multiple fractures, though all the brothers had had fractures half-a-dozen times. None of the brothers was five feet in height. All had scoliosis and kyphosis of the spine. The

youngest brother had two children, both of whom had had fractures. The eldest of the three was the only one who had plugs of wax in his ears.

Sir JAMES DUNDAS-GRANT said he hoped a diagnosis of otosclerosis would not be expressed rashly, as many held it to be equivalent to a death warrant. He had seen many cases in which the expected progression to stone-deafness did not happen, the condition became stationary, and some even seemed to improve. Some of the cases might not have been pure otosclerosis; the same kind of deafness and most of the symptoms of the condition could be produced by catarrhal changes. In the schedule the Eustachian tube condition was omitted. A condition of otosclerosis was that the tubes should be free. If catarrh was present, the case was not one of pure otosclerosis. A hopeless attitude should not be taken up on first seeing the case, and time should often be allowed for trial treatment to have effect before making a pronouncement. He tested after inflation to see whether there was any improvement, and he often placed in the hands of the patient, for self-use, a Politzerizing apparatus; in which the addition of chloroform vapour caused better penetration. In some cases he had had to postpone and even abandon the diagnosis of otosclerosis. The otosclerotic patient was liable, like everybody else, to catarrhal conditions, which were apt to be neglected. Thirty years ago he saw a patient who had the typical features of otosclerosis. She had married, and had assured him (the speaker) that the birth of her children had had no detrimental effect. Recently he had seen her again, and found her deafness worse. He had, however, put her in possession of the inflating instrument to use for herself, and she had now written to say that her friends had noticed an improvement in her hearing.

His own observations supported those of Mr. Jenkins that the conditions in osteitis deformans were different from those in otosclerosis. In his (Sir James's) cases[1] there was narrowing of the Eustachian tubes, in one there was also immobility of the malleus.

Dr. J. S. FRASER (Chairman) said he did not think that inflammation was the "be-all and end-all" of otosclerosis, but it could not be entirely left out of account. Mr. Richard Muir, of Edinburgh, an expert pathologist, when he examined specimens from his (the speaker's) cases of otosclerosis, had concluded that otosclerosis was a chronic inflammatory affection. [Slides shown.] In all the cases from which these slides were taken the patients had suffered from middle-ear disease, and all had demonstrated conditions which, if not otosclerosis, were certainly closely allied to it.

As Professor Mayer had said, otosclerosis was very common. If it was admitted that the six cases, of which he showed specimens, were suffering from otosclerosis it was only right to state that he (the President) had examined two hundred cases histologically. He did not think, however, that 3 per cent. of the population had otosclerosis; Dr. A. Albert Gray had put the frequency at 0·5 per cent. It therefore required a little more than the normal incidence of the disease to explain changes analogous to otosclerosis in six out of two hundred cases examined.

Dr. LOWNDES YATES (in reply) said it was important to attack the problem of the ætiology of otosclerosis by a study of cases in which the signs fulfilled the accepted clinical definition of the disease, because it was clear that what clinicians called ostosclerosis was a condition not yet sufficiently defined. It seemed to him that cases which fulfilled the present clinical and pathological definition were not necessarily examples of the same disease. He had discussed in his paper cases which fell within the clinical definition.

The Use of Temporal Muscle Grafts in Mastoid Operations.

By HAROLD KISCH, F.R.C.S.

THE object of the technique about to be described is to obtain, by one manipulation, obliteration of the cavity left in the mastoid bone at the radical—or partial radical—mastoid operation.

This objective is achieved by inserting into the cavity a "tongue" derived from the temporal muscle.

[1] *Proceedings*, xvi (Sect. Otol.), 27.

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For several years I have been using muscle grafts. In the first cases isolated grafts from the rectus femoris were used. A portion of muscle of suitable size was cut and inserted into the cleansed mastoid cavity. A plastic operation was performed on the meatus and the posterior wound was closed.

Four patients were operated on by this method, but in one case the graft died and had to be removed. The results in the others were good and all wounds were healed in under two months.

The amount of discharge from the meatus in these cases was very small, and was derived from the healing attic and middle ear. No discharge could be sucked from the mastoid region, and this suggested that the plastic operation on the meatus could, with safety, be omitted. The question of the viability of the detached graft was also settled, but, as in one case, death of the graft took place, it seemed advisable to use a graft which could be left attached to its parent. The temporal

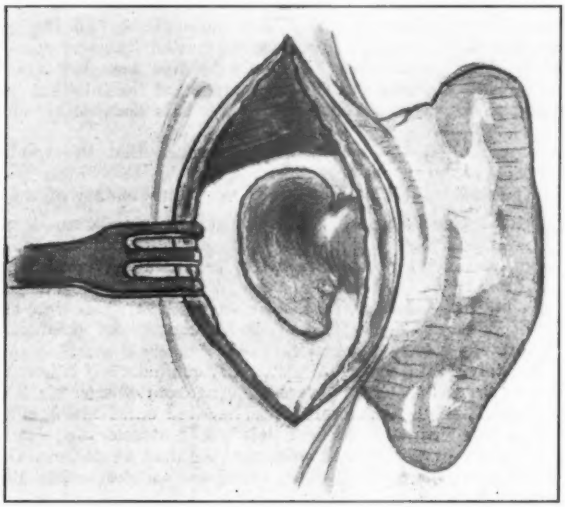

FIG. 1.—Mastoid cavity with the temporal muscle exposed above.

muscle was the obvious one to use. No difficulty was found in exposing, without increasing the incision, a sufficient area of muscle and bringing a tongue of it down to lie snugly in the cavity. The exact technique of the operation is as follows:—The ordinary mastoid operation is performed, care being taken, by using sharp gouges and chisels only, and avoiding the use of burrs and rasps, to avoid unnecessary injury to the bone. The posterior wall of the bony meatus is freely excised, partly to be certain that all diseased cells in this region are removed, and partly so as to obtain subsequently close contact of the graft with the replaced soft meatus. The Eustachian tube is closed by tucking in the mucous membrane to form a plug. Great care is taken not to interfere with the inner wall of the middle ear, as any interference of the stapedial region may result in subsequent loss of hearing. The mastoid cavity is cleansed with peroxide of hydrogen and dried. By retraction and slight dissection a considerable area, at least one square inch in extent, of temporal muscle is exposed (fig. 1) and separated from the underlying bone with an elevator (fig. 2).

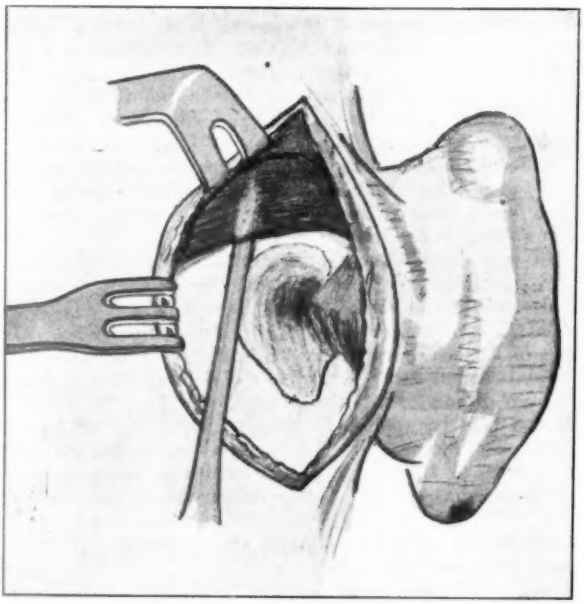

FIG. 2.—Temporal muscles further exposed by retraction. The muscle is elevated and the line of incision seen.

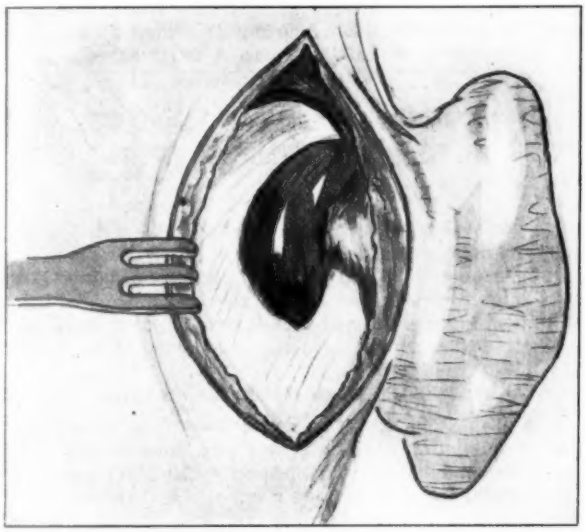

FIG. 3.—The "tongue" of temporal muscle is seen in the mastoid cavity.

With a clean sharp scalpel a tongue of the muscle is cut, half a semilune in shape, and left attached at its anterior end. At the anterior end the fascia over the muscle is carefully divided by a vertical incision. The tongue can then be brought down to some extent towards the cavity, but to enable it to lie snugly in the cavity some of the fibres must be divided, and this is done until the graft lies perfectly, without any tendency to jump out (fig. 3). Care must be taken not to divide the muscle too far forward, or else the temporal vessels may be cut. The meatus is then replaced and the skin incision closed. A plug soaked in paroleine is placed in the meatus. A pad is firmly bandaged over the temporal region to prevent hæmorrhage from any small cut vessel in the muscle.

On the second day the meatal plug is removed, the canal and middle ear are cleansed with peroxide and the plug is replaced. Subsequent treatment consists in syringing with dilute perchloride of mercury solution and the use of peroxide. All after-treatment is painless.

The cut bone of the attic region has to heal, and this takes a variable time. The discharge which comes from it during the healing process is, however, very small and causes no discomfort to the patient.

The method can also be applied to cases in which the middle ear can be left undisturbed except for removal of the incus, as recommended by Mr. Jenkins. In these cases I remove the bridge and extract the incus under direct vision. The graft is then inserted as before. The healing is very rapid, and usually complete in a few days.

The hearing results are good, and all cases are improved.

The advantages of the method are:—

(1) The mastoid cavity is completely filled with living muscle, which attaches itself to the bone, and thus complete obliteration of the cavity is obtained.

(2) The meatus is not divided, so that the after-treatment is painless, and the patient is saved the slow healing of the cut meatal cartilage.

(3) Healing is very rapid. All the radical cases have healed in under two months, and most in six weeks. Even during this time the discomfort is of the slightest as the discharge is so small. In the partial cases healing is complete in a few days.

Fat Grafting in Mastoid Surgery.

By Douglas Guthrie, M.D.

[ABSTRACT.]

An autogenous graft of muscle or fat is frequently used by the general surgeon to fill up a cavity in bone, created by excising a diseased area. Apparently this principle has not been adopted to any extent by the aural surgeon in dealing with mastoid cavities. A brief record of personal experience of the method in ten cases (all of children under 8 years) may be of interest. The cases fall under three headings: (1) Mastoid fistula; (2) chronic middle-ear suppuration; (3) acute mastoiditis.

(1) Two Cases of Mastoid Fistula.

As the result of a mastoid operation there may be a deep depression behind the ear, or even a small fistula leading to a dry lined cavity. It is unsightly, difficult to keep clean, and liable to reinfection. The ideal treatment is to excise the fistula by an elliptical incision, dissect up the edges, remove the lining of the cavity, and implant a piece of fat, from the thigh or buttock, just large enough to raise the skin to the desired level.

(2) Four Cases of Middle-ear Suppuration.

In children, the treatment of those cases of chronic middle-ear suppuration which do not respond to conservative measures is a difficult problem. The results of zinc ionization in small children have not been encouraging. As regards the radical mastoid operation, most otologists will agree that this operation should seldom be performed on children under the age of 10, and never on account of chronic discharge alone, apart from cholesteatoma, polypi, etc. The modified radical operation requires long and careful after-treatment and does not always save the hearing. In a series of eight cases (reported in 1925), discharge was arrested in five cases, but deafness followed or persisted in seven cases.

Post-auricular drainage—that is, the performance of a simple (Schwartze) operation and the retention of a drainage track from the aditus to the surface—is tedious and the aditus and track become readily occluded by granulations.

Probably the best treatment is to leave the tympanum to nature, save for a free enlargement of the perforation, and to deal with the anterior and posterior sources of infection: the anterior by removal of adenoids and applications of argyrol, etc., the posterior by excision (not merely drainage) of antrum and cells, followed by immediate closure of the wound. This method I adopted in ten cases last year. In each case the wound healed by first intention and in six cases the discharge ceased.

In four of the cases obliteration of the cavity was secured by a free graft of fat rather than by blood-clot. In three of those cases the discharge ceased.

(3) Four Cases of Acute Mastoiditis.

A fat graft was introduced into the bone cavity after operation for acute mastoiditis, in four cases. In two of these there was slight redness and œdema of the wound on the third or fourth day, and the graft was removed. In the other two cases the wound healed by first intention, aural discharge ceased, and the patient returned home in a week. Despite those successes, one hardly felt inclined to continue the use of fat grafting in acute mastoiditis, as the "bipp and closure" method gives such excellent results. In twenty-eight operations for acute mastoiditis last year, healing by first intention was secured in twenty-three cases.

In conclusion it may be said that fat grafting in mastoid surgery is an excellent means of reconstruction in cases of mastoid fistula, and a further trial in cases of chronic suppurative otitis in children appears to be justified, but the procedure has no place in the treatment of acute mastoiditis.

Discussion.—Mr. Herbert Tilley said he was glad to find that Dr. Guthrie had had twenty-three out of twenty-eight cases in which there was primary union after immediate closure of the wound in acute mastoid inflammation, a satisfactory result in hospital practice as there was always a great demand on the beds. The method demanded a much shorter time than when one had to wait for the wound to granulate up from the bottom. He had a case last year in which he cleared out the whole of a suppurating mastoid, thoroughly cleansed it, anointed it with bipp and sewed up the wound; this was done on a Thursday afternoon; he did not touch the wound until the following Tuesday morning. When the dressing was then removed, the wound was dry, and the patient left two days later with almost perfect hearing. He wished the method were tried more frequently. He knew there were theoretical objections to it, but so far he had never seen a fatal case following the procedure. He had had no experience of placing fat in the wound, or of Mr. Kisch's operation.

Mr. W. S. Syme said he did not see why fat should not be implanted after acute mastoid conditions if the whole of the diseased area had been removed; that was the crux of the matter of immediate closure.

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Mr. ALEX. R. TWEEDIE said that he was sorry to appear in opposition to the suggestion of filling and closing any septic wound. On one occasion on the removal of a keloid over a mastoid operation wound performed by another surgeon two years previously, he had found the cavity occupied merely with blood-clot, the marks of the gouge still unaltered, and the lateral sinus still exposed with no attempt whatever towards formation of granulation tissue. [Mr. Kisch: I do not graft for acute cases, only for chronic.] He asked what advantage was claimed, as without grafts and with mere cleansing of the cavity with normal saline solution, he expected such mastoid operation wounds to heal in about two months. He was much interested in Mr. Kisch's method of closing the tube by pushing in mucous membrane, and asked if this was invariably successful. As regarded Dr. Guthrie's suggestion of fat-grafting, he gathered that at present this was only suggested, and not urged as a routine treatment.

Mr. C. A. S. RIDOUT said that with regard to acute cases, he was closing about two-thirds of the wound; but he shared Mr. Tweedie's apprehension with regard to a forced closing of a septic cavity.

Mr. T. B. JOBSON said that in these cases the time element was very important; when operation had to be done within from twenty-four to forty-eight hours one had to leave the wound more open than if one had the case at a later stage. In a case which had been "cooked" for ten or fourteen days and in which there was a true abscess underlying the incision, closure by first intention was much more likely than in a very recent case. He had tried Mr. Tilley's method in late cases in which there was true pus, and there had been excellent healing, and some had been completely dry; but in nearly every recent case the wound had had to be re-opened. He regarded it as a question of the development of immunity. When the diseased area was cleared out, the patient overcame the residual sepsis if his blood had developed sufficient immunity.

Mr. J. S. FRASER (Chairman) said that most of the remarks in discussion had been dealing with the after-treatment of acute middle-ear suppuration and mastoiditis, but with that the papers read were not concerned; they dealt with the technique (1) of finishing the radical mastoid operation, and (2) the simple or Schwartze operation.

Mr. G. J. JENKINS said that he very rarely found it necessary to use a muscle-graft in connexion with the radical mastoid operation. When it was necessary he had used the upper part of the sterno-mastoid. Recently, however, he had used temporal muscle in a case in which there had occurred leakage of cerebro-spinal fluid after an abscess of the temporo-sphenoidal lobe had been opened.

Mr. THACKER NEVILLE said he had been using Kahn's skin-flap, which filled the cavity, in addition to plugging the Eustachian tube. After the operation, an incision including skin and fat, was made parallel to the posterior incision in the skin. The flap was then cut at the lower end of the wound. The distal part of the skin which went into the middle ear was pared very thin, so as not to include fat, and this filled up the small cavity if one did not remove the apex of the mastoid, which it was not often necessary to do in chronic mastoid cases. The wound was sutured, and a week later the base of the pedicle-flap was cut; usually a suture was not needed.

Mr. JAMES ADAM said he did what Mr. Kisch recommended in one case, and in ten days all was well and the patient had been sent to a convalescent home. He closed the Eustachian tube by putting in a needle-shaped cautery point at the upper end. He was pleased with the operation, and intended to try it again.

Mr. KISCH (in reply) said he wished specially to emphasize the fact that he did *not* advocate putting a graft into an acute mastoid cavity; to do so would break an important rule of surgery. Free drainage must be provided for acute inflammation. It was not necessary to discuss Mr. Tilley's method, as it did not come into his paper.

In answer to Mr. Tweedie, he did not always succeed in closing the Eustachian tube, but the method described was, on the whole, very successful, and in most cases he was unable afterwards to blow air through the Eustachian tube into the middle ear. He thought anyone who did many mastoid operations would be able to show some cases which had healed within two months without grafts, but he could himself show twelve consecutive cases operated on by the graft method, which were healed in that time. He was certain healing was much more rapid by using this method than by the granulation or skin graft methods.

Dr. GUTHRIE (in reply) said that he would not recommend fat grafting as a routine procedure. In acute mastoiditis he would not recommend it at all, in spite of his two successes. The "bipp and closure" method, in acute mastoiditis, gave results which could hardly be surpassed.

He regretted that no one had discussed those difficult cases of chronic middle-ear suppuration in children which resisted conservative treatment. He had secured good results from the ordinary Schwartze operation, with thorough disinfection of the bone cavity. Instead of packing and repacking until the cavity closed, and instead of permitting the cavity to fill with blood-clot, which might become organized or might remain, as in the case described by Mr. Tweedie, he introduced sufficient fat to fill the cavity, and he stitched the wound. This experiment had produced a good result in three out of four cases, and after further trial it might prove suitable for regular use in selected cases.

OTOLOGICAL SESSION, JUNE 2, 1928.

Chairman—Mr. J. S. FRASER, F.R.C.S.Ed.

Vasomotor Affections of the Internal Ear.

By Professor GEORGES PORTMANN, M.D. (Bordeaux).

VASOMOTOR DISTURBANCES may be considered as among the most pathognomonic and the most important in affections of the internal ear. These vasomotor troubles depend directly on the sympathetic system.

We have recently made a series of experiments on the action of the sympathetic in regard to the labyrinth. These experiments have been either peri-carotid sympathectomy or injections of vasomotor drugs (stimuli or inhibitors of the sympathetic; stimuli or inhibitors of the parasympathetic). Our study has dealt also with the circulatory labyrinthine troubles caused by the compression of the vertebral artery or of the "common carotid artery," or by peripheric vasodilatation (warm bath test).

These different experiments, of which the results agree with those obtained by Terracol (perivascular sympathectomy, section of the cervical sympathetic, injection of suprarenin in the superior cervical ganglion), allow of our bringing forward some conclusions on the vasomotor affections of the internal ear.

For a long time the action of the sympathetic on the labyrinthine circulation, on the one hand, and on the secretion of the endolymphatic fluid on the other, has impressed otologists. It is astonishing that the study of it has not been further developed, for in 1904 Ferreri pointed out certain affections of the internal ear in connexion with some sympathetic lesions and spoke of "Glaucome," a term used again these last years by Aboulker and by myself, to designate the hypertension of the endolymphatic fluid.

Escat, on the other hand, in a very complete work published in 1906, on arteriosclerosis of the labyrinth and of the acoustic centres, has devoted a large part to vascular spasms in the pathogeny of Ménière's disease. But it is especially Lermoyez who (in 1919) has insisted on the vascular labyrinthine phenomena and their clinical manifestations. He showed the importance of angiospasm of the vestibular or cochlear branches of the internal auditory artery in certain cases of deafness or vertigo.

Having observed a typical angiospasm of the anterior labyrinth in a patient affected with Raynaud's syndrome, Lermoyez drew attention to the similarity of structure between the two situations, digital and labyrinthine.

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The stenosis of the internal auditory artery, which is brought about slowly, causes a gradual anæsthesia of the ear, that is to say, deafness. But then the spasm ceases suddenly; the blood rushes again into the labyrinth which is stunned by it. This too sudden afflux, which in the fingers produces ordinary pain, causes here both the special pain of the cochlear organ, which is the buzzing, and the suffering of the vestibular organ which is the vertigo. Besides that, it abolishes the anæsthesia of the ear, i.e., deafness.

And thus Lermoyez has summed up under the name of "the vertigo that makes one hear" a particular labyrinthine affection, which seems to be connected really with a spasm of the internal auditory artery.

It is expedient, from another reason, to oppose this syndrome to Ménière's disease: this sudden paroxysmic apoplectic and recurrent vertigo, which breaks out in the repose of full health, and which was so long united with cerebral apoplectic congestion.

In Ménière's disease, and in Lermoyez's syndrome, the phenomena are the same, but they make their appearance in reverse order.

The vertigo which in one case inaugurates the fit, in the other ends it. In one case it suddenly impairs the health, in the other it restores it; in one case it brings deafness, in the other it cures it; but in both affections it always comes unexpectedly.

A Table for comparison will better explain the opposition of the two syndromes.

Ménière's Disease	*Lermoyez's Syndrome*
Good hearing	Good hearing
Sudden vertigo	Sudden vertigo
Sudden deafness and decreasing	Gradual deafness
Slow recovery	Quick return of the hearing

The angiospasm would very well explain the onset of vertigo and the disappearance of the deafness. These two symptoms being dependent upon the same cause: the removal of the barrier of the labyrinthine artery. These are simultaneous phenomena: hearing does not return because the vertigo sets in, but when the vertigo sets in, this vertigo is not the cause of the return of hearing; it is only the disagreeable witness to its presence.

If one has put in perfect opposition from a clinical point of view Ménière's and Lermoyez's vertigo it has been the same as to pathogeny. One wished to oppose the congestion and even the labyrinthine overflow of the first, to the ischæmia of the second, consecutive to the arterial spasm.

Nothing is more debatable, however, than the pathogeny of Ménière's disease; hypotheses most contradictory and most opposite have been turn by turn in vogue. At the present time it is still an obscure question which does not appear to be near solution.

The first in date of these hypotheses was that of Ménière himself: viz., that the affection was caused by an intra-labyrinthine hæmorrhage; this, he said, had been proved at the necropsy of a young girl who had died after several severe attacks.

Alexander, however, strongly attacks this opinion and advances the hypothesis that it is rather a question of a patient suffering from leukæmia with leukæmic hæmorrhage in the labyrinth.

Quincke, on his side, has stated his opinion that Ménière's disease is due to a phenomenon similar to that which causes the "dermic circumscribed œdema." He thinks no doubt of an acute angioneurotic œdema in the labyrinth, followed by an endolymphatic hypertension.

According to Wittmaack it is a question of intermittent variation of hypertension of the endolymphatic fluid, entailing modifications of the intra-labyrinthine

pressure. This author, relying on his experiments, thinks that the hypertension is due to a hyperplasic catarrhal state of the middle ear, with diffusion of toxic substances through the "windows."

Escat postulated a different pathogeny for each clinical form of Ménière's disease.

(*a*) *The paroxysmic form*, with remission absolutely complete, seems to realize the *intermittent labyrinthine claudication* by spasms of the internal auditory artery. The mechanism of the giddy fit would be then the same as that of Lermoyez's syndrome.

(*b*) *The apoplectic form*, in which the three symptoms reach their apogée from the first "ictus." This postulates the sudden, complete and irremediable loss of hearing. This form has incontestably as its most constant substratum an intra-labyrinthine hæmorrhage.

The rupture of the internal auditory artery is really here the equivalent of the rupture of the artery in cerebral hæmorrhage and of epistaxis.

The Danish authors, S. H. Mygind and Hwidt, are of the opinion that the fits of vertigo are in connexion with an intermittent increase of the endolymphatic pressure, but do not give any indication of the cause of this hypertension.

Other authors, as Bárány, Aboulker, Quix, have thought rather of an intracranial origin. They see in a hypertension in the area of the lateral cisterna, causing a pressure on the eighth pair, and also on the medulla oblongata, the pons and the cerebellum, the cause of Ménière's vertigo.

Then Quix asserts that this hyper-pressure must act above all on the otolithic apparatus by the way of the "ductus endolymphaticus."

S. H. Mygind makes a similar suggestion, while Thornval, who thinks also of an affection of the otolithic system, places the original lesion in the medulla oblongata.

Kobrak ascribes the pathogeny of Ménière's vertigo to angioneurotic fits of the eighth pair of cranial nerves, depending on two principal causes: (1) a great instability of the vago-sympathetic system, and (2) a local labyrinthine trouble.

By experiments with pharmacodynamic substances (atropine, pilocarpine, adrenaline) Kobrak tries to find in each individual case whether it is a question of vagotony or one of sympathicotony.

The examination of these different theories shows the important place given to vasomotor troubles in the pathogeny of Ménière's disease.

Whether it be Quincke, Wittmaack, S. H. Mygind, Hwidt, for whom it is a question of an endolymphatic hypertension, which can but be closely connected with vascular phenomena, or Kobrak who considers the direct cause in the vago-sympathetic system, the vasomotor disorders—vasoconstrictor or vasodilator—seem really to constitute a pathogenic primordial element.

And Vernet, taking as two extremes Lermoyez's ischæmic-vertigo and Ménière's vasodilatation vertigo, thinks that between these two syndromes there is place for a whole series of troubles ranging from ischæmia to congestion.

If with Lermoyez's syndrome there may be a spasm of the internal auditory artery or one of its branches—vestibular or cochlear artery—there may, on the contrary, exist a vasomotor stasis in the territory of this artery or one of its branches.

This congestion will expel the access, as the latter is expelled by the sanguineous irruption which comes to destroy the labyrinthine anæsthesia on Lermoyez's patients

The vasomotor stasis of the vestibular artery may cause a giddy sensation apart from any other manifestation. All this agrees with the opinion that vertigo is essentially a phenomenon of irritation.

All otologists know that vertigo disappears with the destruction of the labyrinth and that an irritation of the internal membranous ear or of the vestibular nerve is necessary to cause the giddy sensation.

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One sees on the other hand the reflex vasodilatation which locally accompanies the least irritation of the tympanum and which, when this irritation is too violent, also produces vertigo.

A strident noise, irritating the original filaments of the cochlear nerve, may also produce vertigo by reflex vasodilatation.

These few examples, joined to the experiment, show well the connexion which exists between vertigo and the phenomena of vasodilatation in the area of the labyrinth.

By what inward mechanism is this irritation of the vestibular nerve produced? Is it simply a disturbance of the vasotonic equilibrium of the capillaries, under the more frequent form of stasis, which acts directly; or is there, as a necessary medium, some modifications of the endolymphatic pressure?

It is not possible to give an explanation of this problem in the present state of our knowledge.

One cannot in all cases state with precision the connexion between the capillary circulation and the endolymphatic pressure. Perhaps one might see in this increase of the endolymphatic pressure a disorder in relation with capillary stasis. It is the same as in œdema in peripheral stasis; and it is that idea which has guided the surgical treatment of certain cases of vertigo: either one acts by decompressive trepanning, or one performs my operation, viz., the opening of the saccus endolymphaticus. On the contrary, is it possible, as Vernet thinks, to consider these cases of endolymphatic hypertension, as causes of the reflex vasodilatation and vertigo at the same time as the sympathetic phenomena associated?

It is difficult to reply in a decided manner, but it is nevertheless true that, whatever the inward mechanism of production of these vertigoes, it seems to be, in Ménière's disease, closely connected with the vasomotor phenomena, and hence with the sympathetic equilibrium.

From this rapid review of the different affections of the labyrinth in which one has brought in, from a pathogenic point of view, the probable existence of vasomotor troubles, one must separate some precise ideas:—

Vertigo, when a sign of vestibular suffering, seems to be caused by reverse vasomotor troubles; for example, it is produced by the sudden vasodilatation which follows the spasm in the syndrome of Lermoyez.

The vertigo which, on the contrary, accompanies a rather pronounced ischæmia of the labyrinth disappears after sympathicectomy, of which the consequence is a vasodilatation of the labyrinth.

Tinnitus, means cochlear suffering and follows apparently the same rules, being produced either by vasoconstriction, or by vasodilatation: it may be cured or ameliorated, according to the case, when one provokes in the region of the labyrinth the vascular action contrary to that which has caused it.

Vestibular reflectivity seems to be precisely proportioned to the vasomotor modifications. To *vasodilatation* corresponds *hypo-excitability*—to *vasoconstriction*, *hyper-excitability*. All experimental tests and all the clinical observations agree on this subject, and this is perhaps the most important and the surest result obtained up to the present in the study of auricular spasms.

Deafness appears to accompany ischæmia of the anterior labyrinth, caused by vasoconstriction. Sometimes it may be ameliorated or disappear by the action of the vasomotor phenomenon, contrary to that which caused it, that is to say the vasodilatation. But if we bear in mind the tenderness of the cochlear sensorial elements we shall understand that a prolonged vascular obstruction may involve this irremediable damage and definitive deafness.

Is it possible to select from these scattered observations a clinical syndrome of the vascular spasms of the internal ear? I think that we may now settle the chief symptoms, reserving the right to modify them in detail when research on the vasomotor mechanism of the labyrinth is more advanced. The arterial spasm provokes in a determined labyrinthine territory, ischæmia, more or less pronounced, which entails signs of suffering of the sensorial apparatus, namely, vertigo and buzzing, functional troubles and vestibular hyper-reflectivity. The term "spasm" implies a close participation of the peri-arterial sympathetic in connexion, in most cases, with the loss of neuro-vegetative equilibrium.

The angio-spasmodic syndrome of the labyrinth will consequently include: (1) Tinnitus. (2) Deafness, vestibular hyper-excitability. (3) Sympathetic hypertony. These symptoms have always been in accord in the various investigations which I have made, and constitute a constant syndrome with certain modifications, due to the particular ground submitted to the vasoconstriction: anterior labyrinth, posterior labyrinth, or both together.

Besides this syndrome of arterial resistance or hypertonicity of the labyrinth, we must place the syndrome of hypotony or laxity, in which we shall meet the classical signs of the sensorial suffering, but with vestibular hypo-excitability and sympathetic hypotony.

These two syndromes may, moreover, succeed each other alternatively in the same patient.

According to the individual, one observes, in fact, different reactions of the vegetative system under the influence of various causes.

But most often one meets with a hypertonic or parasympathetic syndrome, which, as we have seen, permits us to classify individuals into vagotonic and sympathicotonic. One meets in clinics, fairly often, subjects with whom the disequilibrium seems to be caused by a global hyper-excitability of the vegetative nervous system. It is this particular state that authors have described under the different names of: "neurotony" (Guillaume); "total disequilibrium of the whole system" (Laignel-Lavastine); "vegetative dystony" (Sicard); "amphotony" (Danielopolu).

However this vegetative dystony may be in some patients with vagotonic predominance, in others with sympathicotonic predominance, and with certain subjects, as Harvier and Chambrun have pointed out, hypertony predominates over the sympathetic on the level of one organ, and the parasympathetic on the level of another organ.

The result is a truly perplexing instability of visceral innervation.

The most diverse causes will be found as the origin of the vago-sympathetic troubles and hence of the labyrinthine vascular spasms. The causes may be either mechanical, endocranial, toxic, or even simply psychic.

The most important causes susceptible of acting on this regulating apparatus are undoubtedly *the action of the nervous system and the action of the endocrine glands*, above all, the hormone of the suprarenal gland—adrenalin. Is the hypertonicity of the arterial coat during the spasm, due to the hyperactivity of the sympathetic or to a hyperfunction of the suprarenal?

These are, as Dumas has pointed out, questions to which it is impossible to reply in the present state of our knowledge, but it is certain that it is in this direction that we must continue our researches.

Discussion.—Mr. G. J. JENKINS said that in 1914 he had read a paper on vertigo (non-infective), suggesting that some of the symptoms might be due to raised intra-labyrinthine pressure, and proposing that an operation should be performed to relieve that pressure. He had not practised the operation since then. Later, there had been a conjoint discussion on vertigo,

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during which he drew attention to the low blood-pressure in certain people as probably causative of this syndrome. He had never met with labyrinthine symptoms resulting from too high a blood-pressure, but these symptoms were often associated with too low a blood-pressure for the condition of the blood-vessels of the patient. A colleague suffered from attacks of vertigo, and could produce an attack by having a hot bath. He (Mr. Jenkins) saw him once after a hot bath; he was vomiting and had severe vertigo, with occipital headache, tinnitus and some deafness in one ear. While lying flat he appeared to be normal, but the symptoms returned when he sat up. It seemed to him (the speaker) that in this case there was a variation of pressure in a person whose labyrinths were not equal. Deafness was greater in one ear than in the other. He knew of a practitioner with a blood-pressure, normal to him, of 210 mm. When his pressure dropped to 180 mm. he had to go to bed because of nausea, giddiness, headache and tinnitus. That man also had unequal ears. The explanation offered by Professor Portmann was most valuable. Would he also explain how it was that in some of these cases of vertigo, after the attack, the patient heard much better for some time afterwards? This colleague who had attacks of giddiness regularly, always felt better for a week or two after the attack, and also heard better.

Dr. SALISBURY SHARPE said he saw many patients with high blood-pressure who suffered no discomfort and no symptoms while the pressure was even pathologically high, but suffered from vertigo if the pressure was rapidly or greatly either lowered or raised—but especially if it was lowered.

Dr. N. H. PIKE asked whether hypertension could be relieved by injections of hypertonic saline. It was valuable in increased intracranial pressure and in cases of tension in the eyeball, and he wondered whether it would be useful in vestibular cases. Because pressure was high in the radial artery, it did not follow that it was increased in small terminal arteries. It was not so in the eye. He had seen cases with high blood-pressure in the radial, but the pressure in the terminal retinal arteries was lower than normal, and it might be so also in the internal ear. Rectal injection of magnesium sulphate might be used.

Sir JAMES DUNDAS-GRANT referred to experiments on rabbits in which both vertebral arteries were plugged and one common carotid artery tied, and therefore only one common carotid was left. This was experimented upon in connexion with the sympathetic. Stimulation caused contraction, and it was concluded that there was then diminished vascularity of the vestibule. This produced hyposensitiveness of the labyrinth, as shown by lengthening of the time required to induce the caloric reflex. (Demetriades and Spiegel, *Journal of Laryng. and Otol.*, xxxix, 231.) Professor Portmann had said that there was hypersensitiveness; this was contrary to what the other investigator found. He (the speaker) had performed a similar experiment: instead of blocking up the hole through which the vertebral artery passed, he compressed the vertebral arteries in the suboccipital triangles in the depressions behind the mastoid processes. He could compress one common carotid and compress the two vertebral arteries, and found that these produced the same effect. But even when he only compressed the two vertebral arteries, it took longer to produce the caloric reaction than when the pressure was taken away. This procedure, he considered, was of considerable use and easy to carry out.

With regard to hypertension and hypotension, there were cases in which the blood-pressure was low, and giving iodide of potassium made the patient worse. Lake was the first to suggest ergot or ernutin which afforded considerable relief in such cases. By testing with bromide and iodide of potassium one would know in which direction to look for relief. In labyrinthine vertigo, i.e., from unilateral disease in the internal ear, great relief followed small doses of quinine; this could only be ascribed to a direct sedative effect on the vestibular nerve. In unilateral cases the effect was to diminish the excitability on the *sound* side. Often, as Mr. Jenkins had remarked, the vertigo was due to there being inequality between the labyrinths of the two sides.

Mr. A. D. SHARP (Leeds) said there was another type of case in which vertigo and tinnitus were associated with increased blood-pressure, and when means were taken to reduce the blood-pressure there was a relief of the tinnitus.

Mr. THACKER NEVILLE said the improvement of tinnitus by the application of atropine to the tympanum had been demonstrated in the previous year, and was interesting in the light of Professor Portmann's work. Was Professor Portmann willing to do double

sympathectomy? How did he know which was the hyper- and which the hypo-sensitive ear: did he rely on the Babinski-Weil test? If both caloric reactions were weak and the turning test did not give a reaction, how would one decide? He (the speaker) had a deaf patient whose caloric reaction was weak, and the turning tests gave no reaction. On undergoing the Babinski-Weil test sometimes he walked to the right, sometimes to the left. How did Professor Portmann account for this variation and does he recommend double sympathectomy in that case?

Professor PORTMANN (in reply) said he had not always found, in his experiments, a direct relation between the general blood-pressure and the ear trouble. The accounts of the different experiments made could be seen in the respective papers. We might have vertigo either with high blood-pressure or low blood-pressure, as for instance after a severe hæmorrhage. He always found hyperexcitability of the vestibular apparatus during a warm bath, and yet the blood-pressure was going down.

He had read Sir James Dundas-Grant's accounts of the results of compression of the carotid and vertebral arteries; sometimes there was hypotension, sometimes hypertension, and one could not judge which it would be. It was not a sure method. One must always remember the anatomical variation in different individuals. Quinine, he considered, acted as a vasomotor drug. He had injected atropine and adrenalin into rabbits and into human beings, and in many cases they produced a modification of the general circulation, but there were numerous sources of error. A modification of the condition of equilibrium in the sympathetic system could produce opposite syndromes in a few minutes.

The Influenzal Ear.

By W. SALISBURY SHARPE, M.D.

THIS is not a scientific paper, but a retrospect of cases extending over a period of forty years.

At the combined meeting of the Sections of War and Otology, which took place on April 13, 1928, I was much surprised to hear it stated that otitis media, whether suppurative or not, when it occurred as a part of, or as a sequence of influenza, need be little noticed, as recovery from it was usually spontaneous. The experiences quoted below will show that I consider this a very dangerous statement.

Before speaking of the cases just mentioned, it will be well to consider other forms of "influenzal ear," which, being less common, might otherwise be overlooked.

First, I would mention a true nerve deafness, apparently of directly toxic origin and more or less gradual onset, which occurs sometimes in influenza, and resembles that which sometimes occurs in or follows mumps, very severe measles, or other of the specific fevers.

This, in its initial stage, is often thought to be due to quinine, salicylates, or other medicines used during the febrile stage; but this theory is negatived by its tendency to remain permanently, and by its occurrence when no drugs have been employed.

In the course of time many of the patients improve very much, but few recover their former hearing in all its acuteness.

The second class of cases is that in which acute myringitis is present: this may occur without bulging or other evidence of intratympanic tension. Early attention is drawn to it owing to the symptoms of aural pain and dullness of hearing.

The membrane will appear bloodshot, red and thickened, and very generally one or more petechiæ, or small intramembranous hæmorrhages, may be seen. Apart from direct injury, such hæmorrhages (traces of which are visible for some months) are evidence of the influenzal origin of the resulting impairment of hearing.

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During the earlier influenzal epidemics on many occasions I treated these cases by thorough scarification without cutting through the membrane, and afterwards by the application of warmed pure glycerine or glycerine and ichthyol. This was very successful in relieving pain and the hearing was ultimately unimpaired. Such cases need careful watching, as at the first sign of bulging a free myringotomy should be carried out.

It is possible that some of the cases in which hearing is suddenly or very rapidly impaired or lost in influenza may be due to similar small hæmorrhages occurring in or near the auditory nerve.

The next class of case consists of those in which there is inflammation within the tympanic cavity. So far from these being cases of little consequence and of self-recovery, they are, in my experience, characterized by great severity and gravity.

In their prompt, early and energetic treatment lies the hope of the prevention of much deafness and of much mastoid disease, with all its incapacities and dangers. Such treatment is based on the sound surgical principle of the prompt and free release of tension. In no case seen and thus relieved *sufficiently early* has the need for a mastoid operation arisen.

The most acute case I have seen was one in which, in the course of an attack of influenza, pain in the ear came on about 7 or 8 a.m.

I saw the patient (a lady of about 40) at about 9.30 a.m. and found the tympanic membrane reddened, dull and having lost its normal concavity. I did not incise it, but prescribed treatment. About 1 p.m. I saw it again when it was of a dark purplish red colour and markedly bulged. Pain was still present.

The patient, who was a guest in a friend's house, had to be moved to a nursing home for operation and for further treatment. Later in the afternoon a general anæsthetic was given and a free incision made through the membrane. A gush of pus stained with a little blood, following incision, completely filled the meatus (a very wide one) and overflowed slightly into the concha. The pus was swabbed out, antiseptics and a boric fomentation applied, and throughout the stage of healing care was exercised to prevent secondary infection from outside. The principal micro-organism present in the pus was the pneumococcus.

A day or two later pneumonia developed, from which the patient made a good recovery.

The ear healed well, no mastoid symptoms developed and good hearing was regained.

If this case had not been dealt with as it was, a mastoid operation would have been urgently necessary a few hours later.

Another case was that of a boy aged about 8 or 10 who had acute middle-ear trouble which had developed in the course of influenza. Soon after the first onset of pain I found the membrane bulged and at once excised it freely. There was no pus present, but a quantity of clear glairy mucus. This ear "leaked like a tap." Clear mucus, sometimes slightly ropy but never purulent, escaped from it apparently at the rate of some ounces a day during a period of about ten days. It saturated a fairly thick pad of wool and invaded the pillow. Very great care was taken to prevent secondary infection. Eventually this discharge ceased; it never became purulent, the membrane healed, and hearing was fully restored.

The tension caused by this abundant secretion would certainly have caused mastoiditis had it not been adequately relieved early, and there would probably have been a secondary infection if the membrane had been left to burst without attention. I also believe that hearing is rarely, if ever, so completely restored after a natural burst of the membrane as after an incision.

In answer to the possible criticism that I am discussing acute middle-ear inflammation rather than influenza, I would reply that the most acute middle-ear cases I have seen have occurred in the course of influenza. Our difficulty as specialists is that we do not as a rule see these cases in a sufficiently early stage to adopt with success what may be termed preventive treatment. I am aware that

what I have said may be taken by the advocates of that view to prove their thesis, i.e., that the middle-ear complications of influenza tend to spontaneous recovery. It is true that I have only emphasized the occasional severity of such complications; but the great number of cases are neither seen nor energetically treated early enough, and the results are acute and sometimes fulminating mastoiditis, or rupture of the membrane, acute suppuration, secondary infection. Chronic suppuration and other untoward events are so common as not only to be known to all of us, but to be something of a reproach to our section of the profession.

I have had my full share of all these things and my experience only confirms me in emphasizing the importance and, indeed, urgency of early measures.

Discussion.—Mr. G. J. JENKINS said that after a discussion on otitis externa hæmorrhagica, the conclusion was that after influenza there were seen some of the mildest affections of the ear, and also some of the most disastrous. The otitis externa hæmorrhagica often seen during influenza was sometimes confined to the external auditory meatus; the tympanic membrane might be perfect; hearing normal; and no signs of middle-ear affection. The patient might suffer from labyrinthine symptoms without obvious middle-ear symptoms. It was not necessary that there should be actual hæmorrhages present. In some cases of influenzal ear there might be much pain, giddiness, and headache, persisting over some weeks, without any sign of hæmorrhage or of accumulation of fluid in the middle-ear. Therefore the condition had a very broad symptomatology, and Dr. Salisbury Sharpe had done good service by calling attention to it.

Mr. RITCHIE RODGER said he had been through several epidemics of influenza, and what had struck him was the varying severity of those epidemics in regard to the otitis symptoms. In the winter of 1926 there were, in the north, two distinct epidemics, in December and March, and in those there were different types of cases. He, like Dr. Sharpe, made a practice of carrying out early paracentesis in all the middle-ear cases, and in the December epidemic he did this twenty times, and in only two of the cases was the mastoid operation needed. The next epidemic was more generalized and severe, and he did paracentesis thirty-two times and two out of every three cases required the mastoid operation. That might mean the presence of an organism of different virulence in the two epidemics.

Dr. Salisbury Sharpe had been fortunate in the case in which there was a great gush of pus; as one wondered where such a quantity of pus came from; there must have been a collection of pus in the aditus and antrum, as well as in the middle ear, where there was a very small cavity. He had been in the habit of looking upon a large amount of pus as one of the indications of probable mastoid involvement.

Mr. HERBERT TILLEY asked whether other Members adopted a practice which he employed; after incising the membrane, he applied an exhaust speculum until normal blood followed the purulent discharge through the incised membrane. His cases had seemed to do better than when the drum was incised and the septic contents were left to find their own way through the incision.

Sir JAMES DUNDAS-GRANT said that most alarming appearances were sometimes seen in cases of myringitis hæmorrhagica. One could eliminate the possibility of severe inflammation in the middle ear, when there was still good hearing.

Politzer in his text-book (5th German ed., p. 443) mentioned a type of influenzal mastoiditis in which, when the mastoid was opened, there was a large cavity, and if that was cleared out and drained, the discharge from the ear ceased, and there was not even the need for opening the antrum. One would like to ascertain the condition of the antrum. He thought the great flow of pus mentioned by Dr. Salisbury Sharpe must have been due to suppuration in the mastoid cells.

Bezold said that mastoids having one large cavity were more liable to suppurate than those with many septa, as the latter had a larger area of bacterium-killing mucous membrane.

Mr. A. D. SHARP (Leeds) said there was a type of case he saw from time to time which appeared to be a sequel of influenza. Earache and tinnitus were complained of. On examination, one or two pin-head size bright hæmorrhagic spots were present on the tympanic membrane.

Dr. SALISBURY SHARPE (in reply) said that one point he specially stressed had not been dealt with in the comments, namely, the fact that the hæmorrhagic myringitis was pathognomonic of influenza. When cases were brought to him with the petechiæ still present he always attributed the condition to influenza.

He was sorry to hear Mr. Ritchie Rodger speak of the large number of mastoid operations he had had to do after free myringotomy. The only explanation of that, he (Dr. Sharpe) thought, was not the virulence of the organism, but the fact that the myringotomy was not done early enough.

Case of Sudden Bilateral Destruction of Cochlear and Vestibular Senses of Non-Specific Origin.

By T. A. CLARKE, M.B. (introduced by Mr. F. J. CLEMINSON).

M. B., FEMALE, aged 36.

History.—Well until four months ago, except for occasional slight indefinite attacks of giddiness during preceding six months. Four months ago had an attack, lasting three days, of vomiting, with giddiness and tinnitus. A fortnight later had a similar attack, after which she noticed slight deafness in the left ear.

Three months ago woke in the night completely deaf, saying in a loud voice: "I am deaf and dumb." No increase in giddiness then.

Since then she has remained completely deaf; giddiness has diminished and strength is returning.

On Examination (May 2, 1928).—Intelligent; apparently completely deaf; weak and unsteady. Cochlea: tympanic membranes normal; no sound heard by air or bone conduction; galvanic tests elicit no response. Vestibule: very slight spontaneous horizontal nystagmus to both sides. No response to rotational, caloric or galvanic tests. General: unsteady, but no definite Rombergism; no alterations in tone; no asynergy. Ocular movements and fundi: normal. Cerebrospinal fluid and blood: normal. Wassermann reaction (blood and cerebrospinal fluid) negative.

Discussion.—Mr. J. S. FRASER (Chairman) said that, in spite of negative response to the caloric and galvanic tests, he thought this condition might be due to hysteria, and he believed that the patient would recover her hearing. The Section would be interested in a further report.

Mr. A. R. TWEEDIE said he agreed with the Chairman. He had only been able to examine the patient for a few moments, but the impression he obtained was that hysteria could not be excluded.

Dr. LOWNDES YATES said he had found that the patient could hear a tuning-fork placed on the mastoid process. He had then produced another tuning-fork and placed one fork on the mastoid and the other close to the auditory meatus. He had repeated these tests, having previously stopped one of the forks, and the patient said that she heard the tuning-fork when the only fork vibrating was being heard by air. He regarded this as definite evidence that there was a hysterical element in the case.

Sir JAMES DUNDAS-GRANT said one test of the genuineness of deafness was the acquisition of spontaneous lip-reading. This patient made no attempt at lip-reading.

Mr. THACKER NEVILLE said that during the late war a test applied in doubtful cases was to syringe the ear out with ice-cold water until the patient vomited. In such cases the hearing returned if the deafness was functional.

Mr. G. J. JENKINS said this was an important case. It was difficult to understand a negative caloric reaction in a case of hysteria, and he was inclined to think that this was not hysteria or that there was something wrong with the caloric test.

Case of Double Acute Mastoiditis the Post-Operative Symptoms of which Simulated Lateral Sinus Thrombosis.

By H. NORMAN BARNETT, F.R.C.S.

P. J., AGED 8, schoolboy, was admitted to hospital on January 5, 1928, suffering from middle-ear deafness, which had existed since the previous September, and was due to infection from enlarged septic tonsils and adenoids.

Family History.—Father died of consumption contracted in the war, and there was a query as to a brother also having had the same disease.

Personal History.—Measles in young childhood.

Tonsillectomy and adenectomy were performed on January 14, and he was discharged on January 16. He was re-admitted upon the 29th suffering from acute double mastoiditis, and was operated on the same night. Pus was found under pressure on both sides; the bone was extensively diseased and of a cellular type; all cells were removed, including the tip cell, the middle-ear was washed out and a great deal of debris brought away. The wound was then treated with B.I.P. and stitched up. Both lateral sinuses were abnormally situated and were uncovered in the course of operation. The surface of the sinuses was slightly abraded and rather grey in colour.

On the following morning the temperature had fallen from 103·6° F. to 101° F., but it rose again that night and became somewhat oscillating, as will be seen by the temperature charts. [Charts shown.] The boy seemed to be going on favourably for the first twenty-four hours, then gradually became worse. The pulse-rate was irregular and a good deal higher than normal; his general condition did not appear satisfactory; it looked as if a lateral sinus thrombosis had developed; but the difficulty about this case was that it was a double one and there was no indication which side, if any, was affected.

On February 2, the urine, which had been normal, became dark red in colour and was found to contain blood and casts. On February 3, the temperature rose to 106° F., and the boy became desperately ill.

In consultation with the physicians the case was reviewed; I could see no certain indication for operation on the lateral sinuses, since we did not know which side to explore, and I felt that it was not a lateral sinus condition but rather general poisoning. The patient was found, then, to have definite acute nephritis. The urine at this time became very scanty—almost suppressed. On February 4 his temperature suddenly came down, but again rose, and the left lung became affected. The physicians' report on the condition was that there was a large area of consolidation, and owing to the suddenness of the onset and the area involved they thought that this was a condition of embolism rather than pneumonia.

Pain, with limitation of movement, developed in the left leg about February 8, and this persisted for some time and was accompanied by wasting. This leg is still slightly wasted.

Gradual improvement set in under careful nursing and treatment about February 18, and the boy, though very weak, held his own, and very slowly began to improve. He was finally discharged on April 12, and is present at this meeting entirely recovered from all effects of his illness with both mastoids and middle ears soundly healed and functioning perfectly, and general health good.

Conclusions.—The case strongly resembled lateral sinus thrombosis, as indicated by the temperature chart, but the course of events rather showed that there was first of all an infection of the kidney producing acute nephritis, carried by the blood

stream from the original site of infection in the mastoid, and secondarily an infection of the lung, probably by an infected embolism, though from what site this embolism came is not clear.

Discussion.—Mr. HERBERT TILLEY said that eighteen months ago he had referred to this type of case, which was first described by Körner, and afterwards, in his lectures on "Diseases of the Nose and Ear," by the late Professor Luc, of Paris, i.e., cases in which there were symptoms of general septicæmia or pyæmia following a primary but fugitive infection of the middle ear. The case to which he (the speaker) had referred was that of a boy who had acute middle-ear infection with mastoid symptoms. As far as could be seen these cleared up without the mastoid being opened. He saw the boy three weeks after the infection when the tympanic membrane was normal and hearing perfect; there was neither pain nor any other other local symptom of mastoid involvement, and it did not seem justifiable to open the mastoid and search for any residue of the primary infection. But the boy had extreme pain over the pubes and under one trochanter (the latter had been mentioned by Luc in two of his cases); there was an evening rise of temperature suggestive of lateral sinus infection, and shortly afterwards there were pulmonary symptoms indicative of pleural effusion. Septic emboli afterwards appeared in various parts of the body, and he (the speaker) advised against operation. The boy died and there was no post-mortem examination. Mr. Richard Lake had written an article bearing on this subject and entitled it "Aural bacteriæmia." In his (Mr. Tilley's) own case the primary ear symptoms seemed have cleared up, while the more generalized infection declared itself.

Mr. J. S. FRASER (Chairman) asked whether blood-cultures were made in these cases. In aural bacteriæmia the blood-cultures were often positive, more frequently so than in definite septic thrombosis. There was always in these cases the question of thrombosis of the jugular bulb. He would like Mr. Donald Watson to relate his recent experience in this matter.

Mr. DONALD WATSON said he had had an ordinary mastoid case. The child had come through the operation very well, but ten days afterwards there had been rises of temperature, running up to 103° F., without rigors, and with some sweating. He (the speaker) had tried the treatment for puerperal septicæmia with intramuscular quinine and anti-streptococcal serum, but it had not done any good. Six weeks later he had ligated the internal jugular vein. After cutting the vein, he looked at the upper end, and there was a little thrombus filling the upper end of the jugular vein, extending from above. The child had had no more rises of temperature, and had completely recovered. He believed that nearly all cases of supposed aural bacteriæmia were instances of jugular thrombosis.

Dr. JAMES ADAM said that six years ago he had had a pneumococcal epidemic in his wards in the Royal Infirmary, Glasgow, and five deaths had occurred from pneumococcal meningitis. One case had been like Mr. Tilley's, and the patient had recovered with impaired power in the leg. A patient with cerebellar abscess, operated on six weeks before the epidemic began and regarded as cured, suddenly became worse and died. A brother of this patient who had been visiting him was admitted with pneumonia. A patient after a mastoid operation was sent home nearly well, when the wards were cleared, and two days later had a pneumococcal sore throat from which he recovered. The temperatures of every patient in the male and female wards during the epidemic were raised, except that of one child. This child, before the epidemic began, had been several weeks in the ward with pneumococcal pus streaming from eyes, nose and ears. No treatment did any good until pneumococcal phylacogen was given, then all the symptoms cleared up except those in the ears. Mastoid operations were performed. One ear had healed, but the other had not. Several months after the wards had been cleared this child had been readmitted on account of a discharging ear, and within forty-eight hours every temperature in the ward was up.

Mr. NORMAN BARNETT (in reply) said a blood-culture had not been made. The best results had been achieved by saline injections. He had no fixed rule as to stitching up or draining; a number of cases did exceedingly well with B.I.P. and stitching, and healed by first intention; others required draining. He applied B.I.P. and stitched in this case, and

locally all went satisfactorily, the mastoid healing by first intention, though a sinus subsequently developed for a time on one side. He did not think the absence of draining was responsible for complications in this case.

He did not think the lateral sinuses or the bulb of the jugular veins were involved, but thought there was a general infection from the originating site in the mastoid. The case was made more difficult, as well as more interesting, from the fact that it was a double one. The organism was the streptococcus.

Cerebellar Abscess.

By WILLIAM IBBOTSON, F.R.C.S.Ed.

PATIENT, male, aged 19.

History.—Tympanic and mastoid suppuration for many years on left side. When first seen was found to be suffering from intense left occipital headache, no vomiting.

Examination.—Lay in bed on his right side, in a flexed position, moaning in pain. Evidence of very chronic suppuration found in left tympanic cavity; no mastoid tenderness or swelling. No fever, pulse 47, no drowsiness.

Treatment.—Admitted into hospital at once. Further examination of central nervous system and cerebro-spinal fluid, and optic discs did not show any conclusive evidence of intracranial abscess. Radical mastoid drainage performed. Two days later patient showed signs of increasing drowsiness, the headache never having been relieved by the operation, whilst there was now marked increase in tension in the cerebro-spinal fluid.

Operation.—Skull trephined over left cerebellar area, very large abscess, containing extremely foul pus and detritus, evacuated. During this performance respiration entirely ceased for about fifteen minutes, but restarted after injection of several drugs, artificial respiration, etc., the pulse remaining full and steady all the time.

May 14, 1928.—For some weeks past patient has suffered no inconvenience whatever, there being no symptoms or gross pathological signs and the wounds have completely healed.

Discussion.—Mr. N. H. PIKE said that he had recently operated upon a frail woman, who had ceased to breathe during the administration of the anæsthetic. Artificial respiration had failed, and he had carried out a decompression of the temporal region; the natural respiration slowly came back, forty-five minutes after it had ceased. She was taken back to bed and lived three or four hours. At autopsy a cerebellar abscess was found.

In another case a man was admitted with supposed intracranial tumour and respiration suddenly ceased. A team carried out artificial respiration for fifteen hours before the heart finally gave out. At autopsy no tumour was found, but there was internal hydrocephalus, without apparent reason. A decompression performed during the fifteen hours had given no help.

The Otometer. An Instrument designed to Measure Accurately Range and Sensitivity of Hearing.

By O. POPPER, F.R.C.S.Ed.

WHEN electrical means of measurement become available, they eventually supplant all other methods and indeed become the standard by which all others are judged. This holds good when the velocity of high-speed aeroplanes or racing cars is estimated, or when the reaction of blood or the acidity of gastric juice is ascertained, just as high temperatures are measured by the thermocouple. The electrical method in each case rules out the human factor of error.

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In order to estimate the range aud sensitivity of hearing, we require pure notes, the intensity of which can be reduced to a point at which they can either just or just not be heard—the threshold of audibility.

To produce a note of known pitch and to measure its intensity would appear to be a modest demand. In practice this has been a formidable obstacle. Technical details I will omit and merely indicate the principle.

In the instrument shown the source of sound is a special and highly complicated circuit of vacuum tubes—called an oscillator—so arranged as to produce continuously pure tones; these being delivered through a loud speaker. The intensity of these tones can be varied at will and the input of current measured. This input of current at the "threshold of audibility" serves as an index for the patient's hearing capacity for that particular note.

The otometer is calibrated for upward of 1,000 pure notes. (Such a number is, of course, entirely unnecessary.) It is a laboratory instrument, the components of which have been kindly placed at my disposal by the Standard Telephones and Cables, Ltd.

The American audiometer, a similar instrument, has twenty notes—ample for our purpose.

The method of testing hearing is as follows:—

The patient is placed at a measured distance from the centre of the loud speaker —say 5 ft.—with the ear to be tested turned towards it. The other ear is closed. The oscillator is now made to vibrate at thirty-two per second, and the intensity of the note is reduced until the patient just fails to hear it. The current now flowing is measured.

This is repeated for every division or subdivision of the musical octave up to 16,000 vibrations per second.

From the resulting chart it is evident that the lower tones require much more energy to be audible than the higher ones. This may alter our conception of those types of deafness characterized by the loss of the lower notes. Can it be that this energy is consumed in activating the ossicular chain in the case of the lower notes, whereas the higher have no need of this intermediary, but pass straight through? This method of testing hearing may open a wide field for investigation.

Physicists measure sound energy in dynes or units of air pressure per square centimetre. After allowing for the initial inertia of our loud speaker—which has been determined—our readings can be translated readily into terms of absolute units. We have thus a universal language for recording ear tests.

When we consider the fallacies which beset tuning-fork methods, this fact assumes an added significance.

When testing sensitivity with the tuning-fork it is customary to place it "near" (note: the distance is not measured!) the patient's ear. When the patient no longer hears it, the observer places it "near" his own ear, and times the period that he continues to hear it with a stop-watch.

I would like to draw attention to several possibilities of error in this method, with their permutations and combinations.

(1) It is almost impossible to produce two identical forks. Their period of vibration and amplitude varies with the weight and elasticity of the steel used.

(2) The volume of sound at any given point is inversely proportional to the square of the distance from the source of sound. Assuming that the tuning-fork be held 5 mm. in one instance, and 1 cm. in the second, from the external meatus—in the second case only one-quarter of the volume reaches the outer orifice of the external meatus. This is the most common source of inaccuracy.

(3) The observer, by testing against his own hearing, assumes himself to be standard, and holds the fork at varying distances from his own ear!

(4) The element of time is introduced, which is quite alien to the investigation. Time, even as measured by a stop-watch, bears no relation whatever to hearing. We use it merely because it is the only way that we can attempt to calibrate tuning-fork methods. Otometric or audiometric methods discard time as the unit and use energy units, which would appear to be more logical.

In conclusion, I must express my thanks to Mr. Gough and Mr. Osment, of Messrs. Watson and Sons, for placing their laboratory at my disposal, and for their indispensable technical assistance.

Discussion.—Dr. ALBERT A. GRAY said that Mr. Popper had remarked that it was difficult to explain why low notes required so much more energy to become audible than high ones. The reason to him (the speaker) was clear. There was much more energy in a rapid vibration than in a slow one. The energy depended on the square of the velocity of the movement, whereas it only varied directly with the mass. A low note might mean only twenty vibrations a second, whereas with a high-pitched note it might be thousands of vibrations a second, and as the basilar membrane could only vibrate within the limits of small amplitude, much more amplitude of movement was required for low than for high notes to produce audibility.

Dr. LOWNDES YATES said that he agreed that at some future date apparatus which generated sound electrically would be employed for testing hearing. He thought that the practicability of such apparatus was open to considerable question at the moment. He possessed a somewhat similar apparatus and found that the note emitted depended on maintaining constant voltages and on so many other factors that it was difficult to keep the apparatus in order for practical testing, although it was excellent for laboratory work. He believed, however, that even the degree of accuracy obtained by the use of the tuning-fork was greater than that obtainable by the apparatus just demonstrated. For example, the apparatus was obviously difficult to keep in order, and was not in order now, for, in addition to the note which it was claimed was being generated, it emitted a note of about 1,000 double vibrations per second and, as a result, a double noise was going on in testing. In addition to this, the loud speaker used could be heard quite differently in different portions of the room; when opposite to it one could hear the note better than if one was at the side of it, and considerable errors thus resulted. Although electrical apparatus similar to this would ultimately replace the tuning-fork as a means of testing hearing, such replacement could not be carried out just now.

Sir JAMES DUNDAS-GRANT said it was necessary to be practical, and the tuning-fork could not yet be discarded, though one must make allowances for what Mr. Popper said. He (the speaker) had had tested the mode of dying-down of a set of forks with the late Dr. Womack, and had read a paper at the Boston International Congress of Otology, giving the results. The duration time could be converted into figures of actual hearing power. Mr. Popper was trying to provide something which otologists were anxious to test and employ.

Mr. POPPER (in reply) thanked Dr. Gray for his explanation. He agreed that the "hum" referred to by Dr. Lowndes Yates was present at certain frequencies. It could easily be eliminated. It was due to a hasty reassembly of certain components of the instrument rendered necessary owing to the fact that the electricity supply in the room was direct, whereas in the laboratory where the tests were made it was alternating.

He agreed that the tuning-fork could not be scrapped yet; it was adequate for range, but could never be used as a test for sensitivity, because of the sources of error referred to.

The ear during the tests was placed at a distance of 5 ft. from the centre of the loud speaker, from which the maximum intensity of sound issued.

He did not agree with Dr. Lowndes Yates that the instrument was inaccurate. When he began this work he had approached the Staff of the National Physical Laboratory, who referred him for the measurement of sound to the circuit evolved by the Western Electric Company of America, and this was demonstrated. The Western Electric Company had 3,000 persons employed in research, and under the circumstances the purity of the note must be accepted with few reservations.

112 Dundas-Grant: *Perforation of the Tympanic Membrane*

Case of Perforation occupying almost the whole of the Tympanic Membrane, closed by repeated application of Trichloracetic Acid. Cicatricial Membrane still Complete after Ten Years.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, a young woman, when first seen was suffering from chronic suppuration of the middle ear of several years' duration; it was so rebellious to treatment that a radical mastoid operation had been proposed. In addition to the usual antiseptic and alcoholic drops, injections of a weak solution of chloride of zinc were made through the Eustachian tube by means of the catheter, and the discharge was brought to an end. The perforation occupied considerably more than two-thirds of the membrane. After the ear had remained in a dry condition for a considerable time I decided to try and bring about closure of the perforation by means of repeated weekly applications of trichloracetic acid with, however, no very great hope of success. Ultimately complete closure was effected, with great improvement in the hearing power. This has now lasted for at least ten years.

Discussion.—Mr. H. J. BANKS-DAVIS said it was not stated whether the perforation was in the posterior, superior or inferior quadrant, and a point was whether one should leave a perforation alone when an ear was dry. Several times he (the speaker) had tried to close perforations in an ear that was dry, as by applying chromic acid or phenol, but the patients did not hear so well after the closure of a perforation. Trichloracetic acid he regarded as a risky drug to use; it was very corrosive and might erode more of the membrane. This patient said she heard much better, and he was interested to see that on suction the tympanic membrane was translucent-looking, like fine scar tissue. Injection of chloride of zinc through the Eustachian tube was also somewhat risky; he had known acute inflammation of the middle ear follow the procedure.

Mr. HAMBLEN THOMAS said the closing of perforations was very useful, since patients were then less likely to have a re-infection during a cold, or to have attacks of giddiness due to obstruction of the Eustachian tube of the sound ear.

Mr. NORMAN BARNETT said the hearing of the patient was very good, and asked whether this was a special case, or was it usual to get such a good result.

Sir JAMES DUNDAS-GRANT (in reply) said that he put a moistened plug of cotton-wool over the perforation, and he usually found that hearing improved with the closure. He had done this in many cases, but never in one of such a large perforation as this. Trichloracetic acid was a very powerful antiseptic, and it was a very useful application after cauterizing the turbinates, for example. It was recommended by an aurist in New York, and that gave the speaker greater confidence in using it for perforation of membranes. He applied cocaine beforehand, and wiped away any soft crusts adhering to the margin of the perforation, then painted trichloracetic acid round the edges until a white ring resulted. Afterwards he applied bicarbonate of soda to check the further action of the acid, or he might leave it alone. It was important to get the ear dry beforehand. In regard to the present patient, it had been proposed elsewhere that a radical mastoid operation should be done, but he thought it worth while to try and check the discharge by other means. He succeeded by injecting a weak solution of chloride of zinc up the Eustachian tubes. It was interesting to see the arborization of vessels at the edge of the perforation; a film formed and coalesced, and one day the perforation was found to be completely closed. In the first volume of the *Transactions* of the Otological Society there appeared a description of a case he showed.

Case of Vertigo on Suction in a Patient with absence of the Stapes. (Previously shown November 17, 1911.)

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, male, aged 34, was referred to me in 1911 by Dr. Outterson Wood on account of sudden attacks of epileptiform character and dullness of hearing in the left ear. He had been subject to these attacks for about four years and had attacks of vertigo with definite loss of consciousness. I performed ossiculectomy and from that time he was entirely free from the attacks. Either during this operation or subsequently, the stapes appears to have been dislodged and was seen on the upper part of the promontory, the fenestra ovalis being closed by a small membranous diaphragm. From time to time since then Siegel's speculum has been used and it has been noted that during compression little or no disturbance was induced, but on rarefaction most violent disturbance of equilibrium took place, with complicated nystagmus which I was unable to analyse.

In reflection on this case I thought that if such disturbance took place on rarefaction in a person with a complete membrane it would probably be due to fixation of the malleo-incudal joint with loss of the cam action, which only takes place on the incus during compression and not on suction. (See next case.)

Case of Vertigo on Suction in a Patient with Adhesive Processes in the Middle Ear, following Scarlet Fever. Presumably Malleo-Incudal Ankylosis.

By Sir JAMES DUNDAS-GRANT, K.B.E., M.D.

PATIENT, a young woman with undamaged membrane, was recently referred to me on account of extreme deafness of the left ear; the deafness was attributed to scarlet fever in very early childhood. On suction with Siegel's speculum, giddiness was produced accompanied by nystagmus which was, however, not at all definite. It is suggested that this had been due to fixation of the malleo-incudal articulation. By means of repeated suction massage, the joint has apparently become looser as the hearing has improved.

Skiagrams of the Petrous Bone used in Diagnosis.

By DAN MCKENZIE, M.D.

The following case particularly illustrates the value of skiagrams of the petrous in excluding otosclerosis.

The patient, a woman aged 35, suffered from progressive deafness, which began when she was 21. Progress was slow at first, but during the last two years it has been more rapid, especially since she became pregnant three months ago.

The hearing was seriously affected, and the signs were those of nerve-deafness; bone conduction being reduced to minus 15 seconds, and the Rinne was positive in both ears. The highest tone audible was the C. 512 tuning-fork, the monochord not being heard at all. On the other hand, C. 64 was audible by both ears.

Pronounced nerve-deafness in a young person suggested syphilis. But the Wassermann was negative and there was no history of this dyscrasia. As it might have been a cochlear otosclerosis the patient was sent to Dr. Graham Hodgson to have the petrous bones X-rayed.

114 McKenzie: *Skiagrams of Petrous Bone*

Dr. Hodgson reported the films exhibited as showing no evidence of otosclerosis.

The idea of functional or hysterical deafness did not occur to us until the negative X-ray report had been returned.

We then applied the cold caloric test and found the vestibular reactions normal. Not only so, but, as often happens, the shock raised the hearing for the voice from one inch to fifteen feet.

The patient has also become aphonic lately.

The value here of the negative X-ray finding is obvious, but the exhibitor would point out that such a finding strengthens reliance upon positive findings in suspected otosclerosis.

CORRIGENDUM.

Proceedings, xxi, 1775. "Achalasia and Degeneration of Auerbach's Plexus" (Abstract). Owing to a reporter's error the author's name was given as "A. T. Rake, F.R.C.S."—instead of "G. W. Rake, L.R.C.P., M.R.C.S."

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